THE

AMERICAN JOURNAL

OF THE

MEDICAL SCIENCES.

EDITED BY

A. O. J. KELLY, M.D.

NEW SERIES.

VOL. CXXXIII.

QUAE PROSUNT OMNIBUS

15-8333
6/1/21

PHILADELPHIA AND NEW YORK:
LEA BROTHERS & CO.
1907.

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THE

AMERICAN JOURNAL

OF THE MEDICAL SCIENCES.

JANUARY, 1907.

ORIGINAL ARTICLES.

THE PRINCIPLES UNDERLYING THE SURGERY OF THE STOMACH AND ASSOCIATED VISCERA.[1]

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Clinical surgery as applied to the stomach has lacked an accurate physiological and pathological basis. Certain diseased conditions have been relieved successfully, but the reasons for the results have often not been satisfactorily explained. This is especially true in regard to function. In the past we have founded our knowledge largely upon the scientific study of the dead; but this kind of information is grievously open to error, inasmuch as secondary complications and terminal infections frequently obscure the initial lesion, so that from the postmortem evidence we are not always able to get a clear mental picture of the disease during the curable period. The immediate cause of death may be, apparently, disassociated from the primary lesion, which, although completely healed, had set in motion the fatal pathological process. As a foundation, medicine is greatly indebted to postmortem pathology, but as a superstructure, this has a less exalted position, and, too closely followed, leads to inefficiency and pessimism.

Modern surgery has dealt with the practical side and established a "living pathology." It has cleared up those three great avenues of peritoneal infection—the Fallopian tube, the appendix, and the gall-bladder—and is now engaged in a surgical investigation into the diseases of the stomach. The problems to be elucidated, however, are very different from those just mentioned, since infections, primarily, play but a small part in gastric pathology. The gap between the knowledge obtained from postmortem study and the clinical findings must be bridged by animal experiments.

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[1] The Mütter Lecture of the College of Physicians of Philadelphia, delivered December 4, 1906.

In the past we have depended too much upon form without inquiring sufficiently close into function. At present there is an extraordinary interest in physiological experimentation, and its effect upon surgery has been most beneficial. The work of Carrel, Crile, Cushing, and especially Cannon,[1] whose investigations in this field have added so largely to our real knowledge, are conspicuous examples of the trend of recent progress. A vast amount of work in the practical and comparative anatomy of the gastrointestinal tract, has been done by Cunningham, Huntington, McClellan, Deaver, and others, and in the experimental physiology of the digestive system by Pawlow, Starling, and a host of co-workers. It is with the view of bringing together and correlating some of the undigested facts in embryology, anatomy, physiology, and pathology, *which have a bearing on sound surgical practice*, that this paper is written.

I.

The study of the embryology of the gastrointestinal canal is most interesting and instructive and shows how much more permanent is function than form. The primitive intestinal tube is composed of three fundamental parts, the "foregut," the "midgut," and the "hindgut." In form the small intestine begins at the pylorus and the large intestine at the ileocecal valve; but functionally they maintain the rudimentary type, the small intestine begining in the duodenum, just below the common duct, probably at the muscle described by Ochsner,[2] which he has shown is to be found near the juncture of the second and the third portions of the duodenum; and the large intestine begins near the splenic flexure of the colon (Kieth). The ancients, in calling the cecum a second stomach, were much nearer the truth than we have thought.

From the embryological foregut we get the back wall of the pharynx the œsophagus, the stomach, and the duodenum to its third portion, the liver and pancreas being developed from the duodenal end. The gall-bladder and common duct are direct invaginations of duodenal structures (Cunningham). It will be at once noted that all of these organs having their origin in the foregut have to do with the preparation of food for digestion, but are not, themselves, capable of absorption. The stomach and the duodenum have some selective action in absorbing certain things, such as stimulants. In operating upon the upper gastrointestinal tract soon after a meal, I have often been struck with the milky lines made by the full lacteals in the jejunum, while no such appearance was present above this point.

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[1] Amer. Jour. Med. Sci., 1906, cxxxi, 563.

[2] Trans. Amer. Surg. Assoc., 1905.

The duodenum with its wide caliber, fixed position, and its delivery point nearly as high as its pyloric origin, enables the chyme from the stomach to be thoroughly mixed with the pancreatic and biliary juices, absorption taking place below Ochsner's muscle. The jejunum and ileum take up the solid portions of the food, absorbing at least 90 per cent. of the proteids, but by no means do they dispose of all of the fluids. The contents of the ileum at the ileocecal valve are still in the liquid state, though the ingesta have lost the bulk of their nutritive elements. The cecum absorbs the fluids which are held for this purpose between the ileo-cecal apparatus and a physiological muscular contracture (the ceco-colic sphincter) near the hepatic flexure of the colon. In this intestinal segment, the contents are churned back and forth until the fluids have been re-absorbed, and the waste solids driven further along into the transverse colon. It is easily seen that the midgut retains its primitive characteristics; in the jejunum, ileum, cecum, and adjacent colon all of the absorption takes place. Man prepares his food with the organs which have their origin in the foregut, and absorbs his nutrition from the derivatives of the midgut, that is, he eats with the jejunum and the ileum and drinks with the cecum. The posterior "no-loop" gastrojejunostomy does not, therefore, deprive the patient of any appreciable amount of absorbing surface, and the nutrition is in every respect normal. The cecal function explains why cholecystenterostomy into the hepatic flexure of the colon has proved so successful an alleviation in complete obstruction of the common bile duct, when the duodenum for any reason could not be used for the purpose. The continuous bile flow seems to prevent an ascending infection from the colon, although, as compared with the duodenum, it contains quantities of highly virulent organisms. When a permanent external cholecystostomy is made the patient loses with the bile-discharge twenty to thirty ounces of fluid each day, thereby being compelled to drink a larger quantity of liquid, and at best shows signs of dehydration; whereas, when the bile fluids are turned into the cecum they are quickly reabsorbed. The effect of the bile in digestion, especially in aiding the pancreatic ferments, is of course lost, but its usefulness upon the function of the large intestine is preserved.

The value of water in the human economy is testified to by the fact that it is re-distilled in the cecum, and used in the system over and over again. The mechanical effects of fluids in carrying solids as far as the cecum cannot be over-estimated, but beyond this situation liquids would interfere with the storage function.

The primitive hindgut begins near the splenic flexure of the colon, and although largely a matter of convenience, this portion of the intestine has a very considerable nutritive function. The normal action of the upper colon is antiperistaltic, except during defecation. Mr. Bond, in his most admirable address on surgery[1] before the British Medical Association in 1905, showed that particles of indigo-carmine placed inside the anus are carried upward by what he calls "reverse mucous currents." This process occurs close to the intestinal mucous membrane and takes place in spite of bowel passages.

Cannon in his experiments also finds that antiperistalsis is the normal movement in the upper large intestine. By selective action valuable food elements, and especially fluids, are carried back into the cecum. Great advantage has been taken of this physiological fact by the giving of saline infusions by the rectum. Murphy has shown that salines introduced very slowly into the rectum will be absorbed with great rapidity, largely by reverse peristalsis to the cecum. From two to four quarts, which otherwise could not be introduced into the body, except by hypodermoclysis or venous transfusion, can be taken up in this manner. Especially valuable is this in connection with gastric surgery when the stomach is not available and fluids are essential.

Comparative physiology is interesting as showing that in carnivorous animals digestion is practically completed in the small intestine, while in herbivorous animals the colon is equally important, on account of the liquid nature of the plant and grass juices upon which they depend for nourishment. The human species occupies an intermediate position.

II.

The dome of the stomach follows the curve of the diaphragm and rises above the œsophageal opening. In searching with the hand in the stomach for the cardiac orifice, one unconsciously tends to pass above the actual situation, especially as, on irritation, the cardia contracts, leaving the mucous membrane nearly smooth. A little dimple, however, can be found, and steady pressure at this point for a short time, allows the finger to pass through the opening. The lesser curvature has a somewhat fixed position, in the anesthetized patient about two-thirds of its extent hanging nearly longitudinal with the long axis of the body, while the lower, horizontal third, turns sharply to the right and somewhat upward, varying from two to three and a half inches in length. Distention of the stomach comes about, almost entirely, through alterations in the relations of the greater curvature.

The stomach is a contractile organ, exercising its functions through muscular action, gravity playing but a small part in the onward progress of food. When distended it extends downward and to the right, the pylorus being carried across the median line and upward to prevent the weight of the food resting against the pyloric sphincter. In all distended organs having storage function, this elevation of the outlet will be found to prevent continuous exertion of muscular force for retention purposes, as is shown in the urinary and gall-bladders. The stomach is emptied of its contents not only by general contraction, but also through muscular bands which extend downward from the fundus, grasping the greater curvature (Cannon). As these bands contract the greater curvature is shortened and the pyloric outlet brought more nearly to the bottom of the cavity.

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[1] Brit. Med. Jour., 1905, ii, 232.

In a general way it can be said that all of the stomach lying to the left of the longitudinal part of the lesser curvature has storage function (fundus), about four-fifths of the whole (Starling). On the right, having the horizontal part of the lesser curvature as its superior wall, is the antrum, the grinding portion of the stomach. There is said to be a slight thickening of the circular muscular fibers at the entrance to this cavity. Starling states that this is apparent rather than real, and that the antrum is a physiological, and not an anatomical, compartment. By contraction of these circular muscle fibers the food masses are powerfully held in the pyloric end during the kneading process; some prepared chyme escapes through the pylorus, while a much larger amount of food is ejected backward into the fundus which steadily compresses the whole. In pyloric obstruction the antrum loses its identity and becomes a part of the distended fundus, as in the cadaver, accounting for the perfect drainage of a gastrojejunostomy, whereas, if the same operation is done and the pylorus be unobstructed, the artificial stoma fails to drain and is often productive of harm, since the muscular action of the stomach propels the food out through the normal outlet, without regard to the gastroenterostomy.

The terminal three-fourths inch of the stomach next to the pylorus can be classed with the pyloric apparatus, since it has but comparatively little to do with the food grinding, and acts rather as a funnel with the apex at the pyloric ring (the pyloric canal of Jonnesco). The pylorus, under the muscular contractions of the stomach, projects into the duodenum and, from the duodenal side, greatly resembles the vaginal portion of the cervix.

The importance of these muscular features is shown by the fact that nearly 80 per cent. of all ulcers of the stomach are situated in this grinding, pyloric end, the most common variety being the saddle ulcer of the lesser curvature, extending flap-like down the anterior and posterior surfaces (pre-pyloric). The ulcer crater, if one be present, will usually be found upon the posterior wall, or there will be two ulcers facing each other, one anterior and the other posterior, connected superiorly by a bridge of induration.

The pylorus is seldom primarily involved in ulceration, since the pyloric canal is normally contracted, and is less exposed to mechanical injury, or the presence of an excess of acid secretions. The large majority of so-called pyloric ulcers are in reality duodenal. The duodenal ulcer extends up to the pylorus, or within three-fourths inch of it, in 96 per cent. of all the cases; error in accurate localization has led to mistaken identification. As a matter of fact, we find at the operating table that no less than 40 per cent. of all gastric and duodenal ulcers are situated in the duodenum.

On the peritoneal surface of the gastric side of the pyloric ring will be found a peculiar arrangement of the bloodvessels which is rather constant. From the lower side a thick vein passes upward somewhat more than half-way upon the anterior surface. From the upper border a second vein reaches downward in the same line, nearly, if not quite, meeting the first.

Not only is ulceration most frequent in the antrum, but the topography of gastric cancer will be found to be the same as ulcer. In 54 per cent. of 134 resections of the stomach we found malignant disease originating in the submucous tissue at the margin of an ulcer. Primary cancer of the duodenum, however, is rare. We have seen only two instances, although four times we have found carcinoma developing on the gastric side of a duodenal ulcer which had involved the pylorus.

The pyloric antrum occupies a sheltered position under the left lobe of the liver, and this is also true of the duodenum above the common duct. As contrasted with the remainder of the stomach, acute ulcer perforations into the free peritoneal cavity are relatively uncommon in the antrum on account of the ease with which adhesions to the liver, gastrohepatic omentum, the suspensory ligament, and the gall-bladder are formed, and these tend to prevent the gastric contents from escaping. The cardiac end of the stomach has no such protection, and although this region has but 10 per cent. of the total number of ulcers, acute unprotected perforations are here more frequent than in the entire antrum. Perforations of duodenal ulcers are more common than gastric, but the sheltered situation of the duodenum enables ready adhesive protection, while its contents are nearly sterile, and relatively small in amount.

The bloodvessels supplying the stomach are derived from the cœliac axis and are four in number: the gastric, which reaches the stomach on the lesser curvature just below the œsophageal opening; the superior pyloric branch of the hepatic artery at the pylorus; the gastroduodenal, which gives rise to the right gastroepiploic; and the left gastroepiploic, from the splenic artery.

The bloodvessels of the lesser curvature lie in the wall of the stomach; those of the greater curvature are to be found at considerable distance from the gastric wall, arterial branches passing upward from the gastroepiploics upon the stomach, anteriorly and posteriorly in a saw-buck manner. When the stomach is distended, this permits the greater curvature to sag downward toward the bloodvessels without compressing them, enabling rapid change in size and position.

Tortuosity of the uterine arteries makes possible the great size of the uterus during pregnancy, because it comes on slowly, but tortuosity would not allow the rapid changes to which the vessels of the greater curvature of the stomach are subjected. By tying the four bloodvessels, gastrectomy can be made bloodless, just as hysterectomy is in the modern operation.

The lymphatic arrangement is nearly the same as the vascular, but varies in one important particular. On the greater curvature no lymph nodes are to be found to the left of its middle, and the lymphatic circulation of this region is from left to right (Cuneo). Therefore, in radical operations for cancer, it is possible to save a great deal of the greater curvature. On the contrary, in the lesser curvature the lymphatic structures lie in the submucous tissues, rendering it necessary in every case of carcinoma of the pyloric end to remove all of the lesser curvature as high as the gastric artery (Mikulicz). The glands about the pylorus are most numerous on the inferior surface, although several are found just above it in the line of the pyloric artery. The relation of these glands to the field of gastric cancer was long ago dwelt upon by Kocher. The dome, arising above and to the left of the cardiac orifice, is disconnected from the remainder of the stomach in its blood supply and lymphatic supply (Robson and Moynihan). The lymphatics of the stomach, in a general way, greatly resemble those of the other hollow viscera; the neck of organs is the region of the lymphatics, the fundus being less well supplied.

III.

Thanks to the investigations of a large number of experimental physiologists, we know many of the secrets connected with the functions of the stomach and duodenum. One important fact developed is that the stomach not only has many ferments, but that these are called into action by the character of the ingesta, and that they can be increased or diminished by appropriate diet.

Gastric function is carried on to a considerable extent independently of the nerve supply. The chyme when ready to leave the stomach has attained a proper acidity, which causes contraction of the gastric muscles, and the pylorus opens automatically to permit the escape of the chyme. The duodenum also has control over the pylorus, and acidity is here again a feature influencing the pyloric closure. According to Kelling, the chemistry of the duodenal contents is the most important agent in the control of the pylorus. The mucous membrane of the antrum produces specific substances called hormones (Starling), which, acting with nerve impulses, such as sight, taste, and smell, control the amount of food ingested and the necessary gastric secretion.

Not only is much of this process the result of chemistry, but the same force, through the vascular system, stimulates the glandular activity of the liver and pancreas. It has been shown that the introduction of chyme into the duodenum causes pancreatic secretion by means of a product of the intestinal mucous membrane called "secretin," when the pancreas has no connection with the body excepting its blood supply. The great protective agent in preventing self-digestion in the stomach is mucous, and this is apparently true of the entire gastrointestinal canal.

The stomach equalizes the temperature of the ingesta, macerates the food masses in a weak solution of hydrochloric acid and pepsin, and converts the contents into a harmonious whole, the muscular action of the pyloric antrum being the active agent in the latter process. The cardiac end is a temporary storehouse, which enables its possessor to place rapidly a quantity of material where it can be drawn upon as digestion proceeds. To a large extent, then, the stomach is a convenience and obviates the necessity of continuous feeding, just as the urinary bladder and the large intestine beyond the splenic flexure are conveniences to prevent continuous elimination.

Excess or changed secretions, especially acidity, seem to lie behind much of the pathology of the stomach, accounting largely for ulcer in the pyloric antrum and duodenum above the common duct, with its alkaline secretions. Trauma plays an important part in the production of ulcer. We have but to remember the frequency of this malady in the grinding pyloric end of the stomach, and to note that duodenal ulcer usually *originates at the point* which receives the impact of the chyme *forcibly ejected* from the pylorus, to appreciate the influence of local injury in gastric disease.

It has been supposed that gastrojejunostomy would pass the food too quickly from the stomach into the intestine. This fear, however, has been proved groundless, since the stomach does not contract in such a manner as to discharge its contents until the proper chemical conditions have been reached, and the food is ground in the antrum before propulsion is begun.

It is altogether probable that modern methods of food preparation have greatly changed gastric digestion, and that a considerable share of the now very prevalent diseases of the stomach is due to modern dietary changes from primitive conditions, just as the loss of necessity for the grinding action of the teeth has resulted in their premature decay. In aboriginal races many diseases of civilization, such as appendicitis, gallstones, ulcer, and cancer, seem to be rare (Senn).

The nerve supply of the stomach is of two kinds, the vagus and the sympathetic. During fetal rotation, the stomach turns upon its right side, which thereby becomes the posterior wall, and the left vagus lies anteriorly. Terminal filaments of the vagi join with the sympathetic fibers from the abdominal ganglion and form the plexuses of Auerbach and Meissner, which lie in the gastrointestinal wall.

The control of the cerebrospinal nerves over the stomach is limited and has to do with food requirements, but, as far as actual digestion is concerned, the sympathetic is the controlling factor. Beyond the stomach the cerebrospinal system is even less influential, until the sigmoid and the rectum are reached, where again conscious control is essential.

The sympathetic nervous system, developed from mesoblastic tissue, is undoubtedly the primitive one. It is closely allied with the control that is inherent in the gastro-intestinal muscles, and which is myogenic in origin, and of the same nature as His' heart muscle band which regulates the heart-beat. The action of the gastrointestinal canal is regulated largely by this mysterious myogenic force.

The sympathetic nervous system has retained, to a large extent, its primitive control over those organs which have to do with the maintenance of the body, but it is altogether probable that it is losing its prominence on account of the overshadowing development of the cerebrospinal system, and, just as other vestiginal organs, like the gall-bladder, appendix, and wisdom teeth, seem to develop a tendency to disease, so possibly does this mysterious nerve body. It is within the realms of possibility that abdominal ptosis and many forms of neurasthenia are characteristic expressions of evolutionary instability of the sympathetic nervous system.

The one essential difference between the sympathetic and the cerebrospinal system is that the sympathetic is not segmented, and is, therefore, unable to prevent general disturbances upon irritation of any of its component parts. This is especially interesting in connection with certain diseases of the stomach which are exceedingly misleading and which apparently have a common origin, such as pyloric spasm, atonic dilatation, prolapse of the stomach, and gastric neuroses. Now that surgery of the stomach is occupying such an important place, it is vital that we should eliminate these conditions from the operating-room.

We have had a surgical opportunity in a considerable number of cases to examine the stomach during that contraction of the pyloric canal and antrum which constitutes "pyloric spasm." This muscular contraction, when present, causes the patient to experience a sensation which he speaks of as gas pain. It can be observed in operative examination under local anesthesia. The peculiar appearance of the stomach during pyloric spasm may give rise to the belief that physical disease exists, because of a roughened, puckered appearance, which lasts a fraction of a minute, and then changes position or develops a rhythmic contraction, confined to the antrum.

In the most extreme degrees we have found it in connection with stones in the appendix, impacted gallstones, tuberculosis of the intestine, and chronic intestinal obstructions of various kinds. Clinically, it would appear that irritation of any part of the gastrointestinal canal and allied organs, the liver and pancreas, may produce pyloric spasm, and that this condition may overshadow the local disease. Cannon in his experimental work found that after severe injury to the intestinal wall the pylorus became closed and remained so for several hours. It would appear that control of the pyloric sphincter is vested not only in the stomach and duodenum, but also in all of the derivatives of the midgut, including the cecum, and that pyloric spasm may and often does originate in the attempt to prevent food entering the irritated intestinal tract, or, in some instances, a perversion of the physiological function. The greatest care is necessary in differentiating these cases from gastric ulcer and other inflammatory infections.

While it is possible that pyloric spasm may exist as the result of a mucous ulcer, as believed by Eiselberg and Doyen, such is not our conviction, although spasm will mimic gastric ulcer clinically and is often diagnosticated as such. We are inclined to look with a great deal of suspicion upon any supposed ulcer of the stomach which cannot be absolutely demonstrated, not only to the operator, but to onlookers as well.

IV.

The contents of the fasting stomach are relatively sterile; this is equally true at the height of digestion, the chyme, when discharged into the duodenum, being nearly free from pathogenic organisms. This is due largely to the acid gastric secretion; while not actively germicidal, the general influence of all of the secretions of the stomach are against germ life. Cooking renders a large amount of the food consumed sterile, but during mastication many organisms are picked up from the mouth, which teems with bacteria of all kinds (Harrington). Cushing found that with sterilized food the discharge from a jejunal fistula was full of micro-organisms (providing the mouth was kept in good condition), and recommended, in addition to sterilizing the food, antiseptic oral washes and careful cleansing of the teeth previous to operations upon the gastrointestinal tract. The vigorous use of the tooth-brush to those who are not accustomed to it may start up a gingivitis at the end of a few days and temporarily increase the virulence of the organisms. Such oral antisepsis in this class of patients should begin at least two weeks prior to the operation. As the average patient cannot be kept in preparation such a length of time the practical importance of this step in the individuals just referred to is not great. A moderate number of bacteria, particularly bacilli, pass with the food into the duodenum and members of the colon group work upward from below.

Adami and Ford have shown that leukocytes pass out upon the free mucous surface of the duodenum and upper jejunum, picking up particles of fat and micro-organisms of various kinds, particularly bacilli, and by phagocytosis destroy them in the neighboring lymphatics. Some bacteria, however, are continuously carried to the liver and are there annihilated, the pigments of the slaughtered organisms giving rise to the little pigmented areas sometimes found in the liver. A varying number of bacteria, however, are passed through the liver and excreted with the biliary secretion. The bile, therefore, must be looked upon as always infected and it is probably this attenuated infection which gives rise to gallstone disease (Lartigau).

The relative sterility of the stomach and duodenum and the upper jejunum shows why gunshot wounds of this locality have been so much more often followed by recovery when operated upon than those farther down in the intestinal tract.

One interesting feature in this connection concerns the reason why gallstone disease is so much more frequent in women than men. In 1700 of these patients upon whom my brother and I have operated, three-fourths have been women, while just the opposite has been true of duodenal ulcer. In 209 operated cases of the latter disease 73 per cent. were males, and only 27 per cent. females. Granting that biliary infection is equal in men and women, is this sex disproportion due to different mechanical conditions? We have been investigating during life a considerable number of patients with a view to determining whether there is a difference in the arrangement of the duodenum and the common duct, which favored ascending gall-bladder infection in women, and, also, as to whether the mechanics in men were unfavorable for permitting the alkaline discharge from the common duct quickly to neutralize that excessive acidity of the chyme which seems to lie behind the etiology of duodenal ulcer. We have not been able to satisfy ourselves that there is sufficient anatomical difference to explain the phenomena. In the living male subject the first four inches of the duodenum has a higher upward curve than in the female, in whom the corresponding portion is more nearly horizontal—which may have some bearing on the question. There is no question that bacteria can, under certain circumstances, travel up the common duct into the gall-bladder. Bond found that indigo-carmine placed within the anus could be detected in the gall-bladder in about forty-eight hours. It is probable that this sex difference in gallstone disease is physiological, rather than anatomical, and possibly due in some way to the potential capacity of the female liver to care for mother and child.

As one passes down the intestinal canal, bacteria increase in numbers and in virulence, and, in the large intestine, bacterial growth adds considerably to the bulk of the stool. The acid change which takes place in the large intestine is probably caused by germ life rather than intestinal secretions, since the large intestine, when entirely free from feces, has, like the small intestine, an alkaline reaction (Bond).

The problem of securing asepsis during operations upon the upper abdomen is most interesting. To one who reads about the gastric surgery of five and ten years ago, the frequency of fatal results from pneumonia following operations upon the stomach is most noticeable; it had various explanations, the anesthetic and the aspiration of gastric contents regurgitated into the œsophagus, being the most generally accepted hypothesis. In Mikulicz's clinic it was brought out that part of the venous blood from the stomach, instead of passing through the portal vein so that the liver might sterilize it, returned directly through the vascular anastomoses about the cardiac orifice and that this unsterilized blood was the cause of a pneumonia which was embolic in character.

Muscatello demonstrated that the endothelial serous lining of the diaphragmatic area is exceedingly active in absorption, and, based upon this, Clark elevated the foot of the bed after abdominal operations to insure rapid absorption of septic products and prevent peritonitis. The unshed blood in the vessels has no germicidal properties and this septic material when absorbed before being acted upon by the proper tissues sometimes caused general sepsis or was carried to the lungs, producing embolic pneumonia of the same variety that had been noted after operations on the stomach. Fowler pointed out that the inherited resistance to peritonitis in the pelvis, which had been brought about by tubal diseases in women and appendicitis in both sexes, caused absorption in this locality to be slow and therefore advised that, in peritoneal sepsis, the head of the patient's bed should be raised and a drainage tube introduced into the pelvis to drain away the discharges. In connection with general septic peritonitis, we followed the Fowler method, excepting that we raised the patient's head and chest much higher. The betterment in the mortality in peritonitis, actual or impending, was remarkable, although it was soon noticed that, although the patient did very well, there was often no drainage from the tube, therefore drainage was not the sole factor. With the introduction of the holding-clamps during gastric operations, which help so much to prevent accidental soiling, and the raising of the patient to the semi-sitting posture, for a few days subsequently, lung complications have nearly disappeared.

In work upon the upper abdomen on the left side any fluids at once gravitate toward the diaphragm; on the right, they are preented from doing so by the liver. To prevent rapid absorption of these unsterilized products, the raising of the diaphragmatic area, so as to drain this material into the pelvis, is of the first importance, as here by means of the omentum and pelvic colon any septic particles can be rendered harmless by absorption through the lymphatics and radicals of the portal vein. So far as these organs which lie within the active radius of the portal vein are concerned, the liver must be looked upon as an adjuvant to the lymphatic glands in the destruction of micro-organisms.

The omentum, according to Dickinson, Buxton, and Torrey, is a most important agent in developing phagocytosis and opsonins, its germinating endothelium is constantly producing lymphocytes and is capable under proper stimulation of throwing both newly formed phagocytes and those called from a distance into germicidal action. This wiping process is greatly aided by the vermicular and swaying movements of the intestines, which, in spite of gravity, bring all parts of the small intestinal wall in contact with the omentum, the epiploic tags having the same function for the more fixed large intestine.

Dudgeon and Sargent have shown that from some source Staphylococcus albus first appears in peritoneal sepsis and rapidly produces a mild infection; this furnishes the necessary stimulus to the protective endothelium, so that the phagocytes are attracted in time to destroy the more virulent, but later developed, bacteria. Bond has demonstrated that the plastic exudate produced by Staphylococcus albus protects the weakened endothelium and prevents the micro-organisms passing directly into the blood stream. When absorbed by sound endothelium, bacteria are carried into the lymphatics and destroyed.

In closing let me quote from Dr. W. H. Welch's recent address on the occasion of the dedication of the new buildings of the Harvard Medical School: "There is a highly significant and hopeful scientific movement in internal medicine and surgery today, characterized by the establishment of laboratories for clinical research, by the application of refined physical, chemical, and biological methods to the problems of diagnosis and therapy, and by the scientific investigations along broad lines of the special problems furnished by the living patient."

THE PARAVERTEBRAL TRIANGLE OF DULNESS IN PLEURAL EFFUSION (GROCCO'S SIGN).

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In March, 1902, Grocco,[1] of Florence, described, in the following words, a new physical sign which he had observed in pleural effusions: "*Paravertebral triangle of the side opposite that of the pleural effusion.* When, with a pleural effusion of sufficient size, one percusses from above downward, along the spinous processes of the vertebræ, with the patient in the sitting posture, there appears, at the level of the fluid, a dulness which, relative at first, becomes absolute as one passes downward, in association with a progressively increasing sense of resistance. In like manner, by percussing downward on the healthy side, along lines parallel to the spinous processes, there is noted, opposite the dulness in the median line, a paravertebral area of deficient resonance, of triangular shape. One side of this dull area is represented by the line of the spinous processes; another, by the lower border of the area of thoracic resonance for a short distance which varies in length from two to three or more centimeters; the outer side is represented by a line which, starting from the base, rises obliquely to unite at an acute angle with the median line at about the upper limit of dulness. In a right-sided effusion, other things being equal, the paravertebral triangle has seemed to me more marked. Although symptomatology abounds in methods for differential diagnosis between pleural effusion and pulmonary infiltration, there can be no doubt that the sign which I have mentioned may be of value in some cases, especially in right-sided and encapsulated exudates. I shall return to this subject later with a detailed description which may illustrate that which, if I be not mistaken, is a diagnostic sign of pleural effusions hitherto undescribed and worthy of consideration."

Later in the same year, Grocco[2] exhibited two radiograms, one from a living patient, and one from a cadaver in which the chest had been filled with a solution of acetate of lead, demonstrating the fact that the shadow extended past the median line to an extent sufficient to account for the symptoms observed. Koryani,[3] in a study of auscultatory percussion published shortly after Grocco's communication, noted that dulness in pleural effusions crosses the median line, extending over to the healthy side in such a manner as to agree essentially with the descriptions of Grocco. Following this, various clinical observers have given testimony to the diagnostic value of this sign: Silvestrini,[4] Maragliano,[5] Flora,[6] Pieraccini,[7] Bucco,[8] Berghinz,[9] Ferranini,[10] Baduel and Siciliano,[11] Cecchini,[12] Raucfuss,[13] Kozlovsky,[14] Kisel,[15] Kernig,[16] Gracheff,[17] Plessi,[18] and Ewart.[19] Barbieri,[20] on the other hand, on the basis of two cases in which he was unable to demonstrate the sign, and of several experiments on the cadaver in which he was unable, by intrapleural injections, to bring about marked displacement of the mediastinum, concludes that the sign can have no relation to the conditions on the diseased side. Ghelfi[21] also considers the sign uncertain and of obscure origin; he observed it in six cases only, out of thirteen. Rodrigues Alves[22] concludes that the sign is of no value, as modifications in the percussion sound of any sort in one-half of the back may cause a certain amount of change of the same character on the healthy side, in a strip along the spine which he considers somewhat analogous to the paravertebral triangle. This he finds especially true in pneumonia. His plates, however, seem to indicate that he has failed to grasp the essential character of this sign. Gracheff finds paravertebral dulness on the healthy side a constant and valuable diagnostic sign. In some instances, however, he finds that the area is not triangular, but of a hemiovoid shape, the transverse measurement being greatest toward the upper part. He insists, especially, as does Signorelli,[23] on the importance of percussion of the spine. The paravertebral dulness, he thinks, may be simulated at times by changes at the base of the lung on the opposite side, particularly in pneumonia, but *flatness over the spine is distinctive of effusion.*

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[1] Brevi note di semeiotica fisica. Riv. crit. di clin. med., Firenze, 1902, iii, 274–280.

[2] Triangolo paravertebrale opposto nella pleurite essudativa. Lavori di Cong. di med. int. (1902), Roma, 1903, 190.

[3] Ein Beitrag zur differentialen Diagnostik pleuritischen Ergüsse. Wien. klin. Rundschau, 1902, xvi, 300.

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[4] Discussion of Grocco's communication. Lavori di Cong. di med. int. (1902), Roma, 1903, 190.

[5] Contributo alla semeiotica dei versamentipleurici. Boll. d soc. Eustach., Camerino, 1903, i, 18–27; Alcune forme di pleurite. Cron. d. clin. med. di Genova, 1903, ix, 145–149.

[6] Recenti noti semeiologiche sulle pleuriti. Riv. crit. d. clin. med., Firenze, 1902, iii, 407.

[7] Le polmonite massiva o pneumo-bronchite fibrinosa. Clin. mod., Pisa., 1903, ix, 5–8; Il suono retrosternale di Banti, ed il triangolo paravertebrale di Grocco. Clin. mod., Pisa, 1903, ix, 303–307.

[8] Il sintoma di Grocco: il triangolo paravertebrale opposto nella pleurite essudativa. Cronica della clin. med. del Prof. De Renzi in Napoli., N. Riv. clin. terap., Napoli, 1903, vi. 342.

[9] Sul triangolo paravertebrale di Grocco. Riv. crit. di clin. med., Firenze, 1904, v, 530.

[10] Il triangolo paravertebrale opposto di Grocco nei versament ipleurici unilateral ilibri. Riforma, Palermo-Napoli, 1904, xx, 953–955.

[11] Il traingolo paravertebrale di Grocco. Riv. crit. di clin. med., Firenze, 1904, v, 5:21; 37.

[12] Contributo allo studio del sintoma di Grocco, con speciale riguardo alla pleuriti sierosa o siero-fibrinosa destra. Riv. crit. di clin.-med., Firenze, 1904, v, 765–770.

[13] On paradoxical phenomena in percussion of the chest. Russky Vrach, S. Peterb., 1904, iii, 295–6; Die paravertebrale Dämpfung auf der gesunden Brustseite bei Pleuraergüssen Verhandl. d. Versamml. d. Gesellsch. f. Kinderk. Deutsch. Natur u. Aerzte, 1904, Wiesb., 1905, xvi, 202–211, 1 pl. We have been unable to consult the original of this publication.

[14] Discussion of Rauchfuss' communication. Russky Vrach, 1904, iii, 296.

[15] Ibid.

[16] Ibid.

[17] Paravertebral dulness in pleuritic effusions and percussion of the spine. Kazan med. J., 1903, v, 39–51.

[18] Sul triangolo paravertebrale opposto di Grocco. Riv. crit. di. clin. med., Firenze, 1905, vi, 411–414.

[19] The practical value of Grocco's paravertebral triangle. Lancet, 1905, ii, 216; A rapid method for the diagnosis and estimation of pleural effusion. Polyclin., Lond., 1906, x, 13–16.

[20] Sul triangolo paravertebrale: Rendic. d. Ass. med. chir. di Parma, Roma, 1903, iv, 60.

[21] Sul triangolo paravertebrale di Grocco (contributo clinico). Parma, 1904. A. Bartoli, 16 p. 8o.

[22] Do triangolo de Grocco, seu valor. semeiologico e sua semeiogenesis. Rio. de Jan., 1905, R. Braga. 79 p., 16 pl., 4o.

Rauchfuss, who maintains that he has been familiar with the sign for over twenty years, regards it as pathognomonic, although he admits that in some massive pneumonias he has noticed a slight, though rather indefinite dulness on the opposite side of the spine. In the several cases, however, in which such an observation was made there was no necropsy to prove the absence of fluid. Koslovsky, who recognizes the diagnostic value of the paravertebral triangle, has also seen a slight indefinite dulness on the side opposite a pneumonia. Rauchfuss observes that the hypothenuse of the triangle has sometimes a slight outward convexity.

Pieraccini, in a case of pneumothorax with a very small amount of fluid, noticed a clearer tympanitic note on the healthy side in an area analogous to that of the paravertebral triangle of dulness in pleural effusions.

Cecchini noted that, with small effusions, the area of dulness varied greatly according to whether the patient lay on the side affected, when the triangle almost disappeared, or sat up, when it became more marked. With large effusions, the variations with changes of position were slight. Plessi studied the auscultatory signs in the paravertebral triangle and observed that there is usually a marked diminution in the intensity of the respiratory murmur, becoming more evident as one approaches the spine. In one case he found that the characteristic tubular respiration heard over the effusion passed the spine, extending about to the external border of the area, so that it was possible to make out the triangle by auscultation. He also asserts that the coin sound, transmitted characteristically through a pleural effusion as a clear, metallic ring, is also heard in the same manner throughout the triangular area, as well as bronchophony and egophony. Finally, he observed that toward the outer part of the triangle, crepitant or fine moist rales were often heard, evidence, probably, of compressed lung.

With regard to the *cause* of the phenomenon, the studies of Baduel and Siciliano are especially important. Endeavoring to reproduce, experimentally, the conditions existing during life, they filled the abdominal cavity with gelatin in such a manner as to support the diaphragm in its normal position. They then injected the vessels through the carotid. After this, fluid was introduced slowly into the pleural cavity while the subject was in a sitting posture. By this procedure a characteristic paravertebral triangle was produced. On opening the chest on the opposite side they were able to confirm the existence of increased pressure, as the lung tended to protrude from the wound as a hernia, while the finger could readily detect the displacement of the contents of the mediastinum—aorta, azygos vein, œsophagus, heart. In other cases, injecting the pleural cavity with gelatin and filling the gastro-intestinal tract with 10 per cent. formalin, they removed the extremities and the skin, and, after making numerous incisions to allow the entrance of the fluid into the tissues, immersed the trunk in a 10 per cent. formalin bath. After a sufficient number of days, transverse sections were made which illustrated with great clearness the extent to which the pleural accumulation itself extends about and over the bodies of the vertebræ, pushing before it the contents of the mediastinum. The accompanying Figs. 1, 2, and 3 illustrate clearly the changes in relations following intrapleural injections of gelatin. Fig. 1 is taken from Braune's *Atlas*, while Figs. 2 and 3 are from the article of Baduel and Siciliano. All sections were made at the level of the nipple.

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[23] Di un nuovo metodo d' indagine diagnostica; la percussione della colonna vertebrale. Policlin., Roma, 1902–3, ix, sez. prat., 1345–1348.

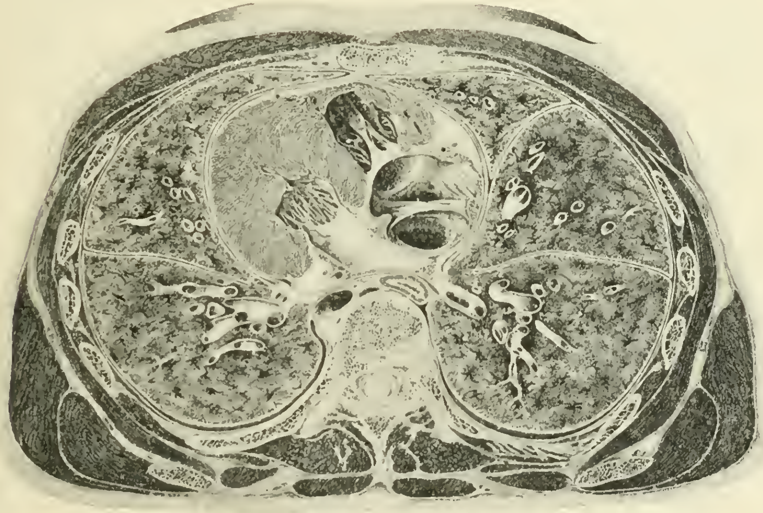

Fig. 1.—Transverse section at the level of the nipples. (From Braune's Atlas of Topographical Anatomy.)

Comparing the clinical data with the conditions as illustrated by these experiments Baduel and Siciliano come to the following conclusions as to the cause of the sign: Percussion of the vertebral column normally gives a clear note because of the contiguity of the pulmonary parenchyma. This note becomes flat at the lower limit of the lungs. In pleural effusion, however, the fluid not only takes

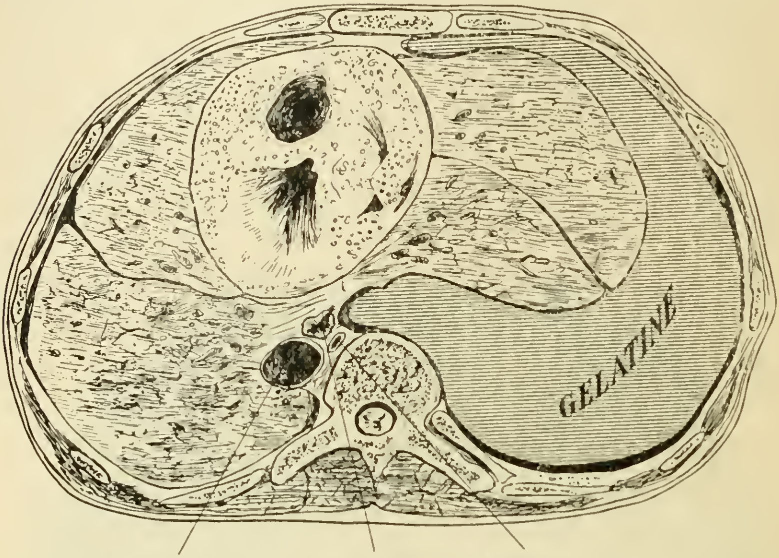

FIG. 2.—Transverse section at the same level as in Fig. 1 in a case of artificially produced right pleural effusion, showing the displacement of the mediastinum and the extension of the pleural contents over the spine. (From Baduel and Siciliano.)

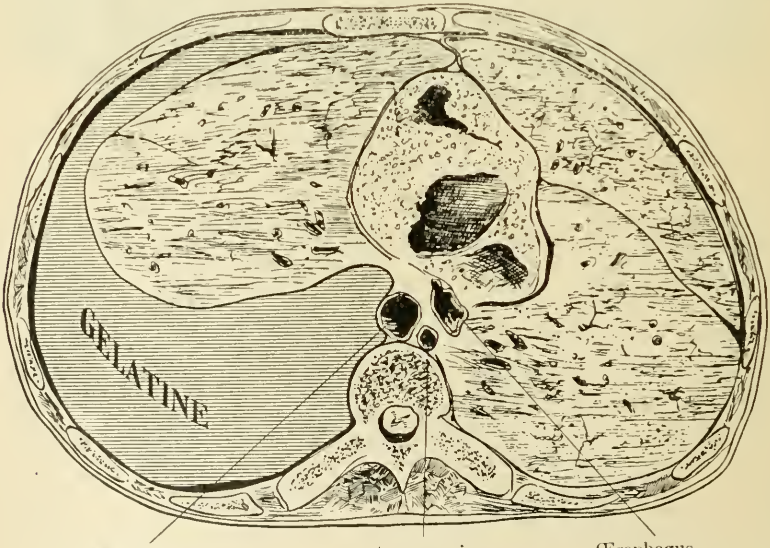

FIG. 3.—Section at the same level as in Figs. 1 and 2 in a case of artificially produced left-sided effusion. (From Baduel and Siciliano.)

the place of one lung, but it extends also anteriorly over the body of the vertebræ, thus inhibiting its capacity for sonorous vibration—that is, it acts as a mute. This dulness extends naturally to a certain extent to the paravertebral region (transverse processes, ribs), especially in the lower part of the chest, where the fluid collects in greater quantity and exercises its action more markedly. In addition to this, Baduel and Siciliano believe that, especially on the left, that is the right-sided effusions, the displacement of the contents of the mediastinum—aorta, œsophagus, azygos vein, and heart—plays a certain part, especially in bringing about a compression of the lung.

Ewart has recently added his testimony to that of the other clinicians already mentioned, as to the importance of this sign. He lays especial stress on the diagnostic value of the variability in the size of the paravertebral triangle with change of position, that is, its diminution or disappearance when the patient lies on the affected side.

Since his first acquaintance with the descriptions of this sign—some two or three years ago—one of us (Thayer) has determined its presence in most cases of pleural effusion which have come to his notice. As time has gone by he has been more and more impressed with the constancy of the sign, the ease with which it may be demonstrated, and its real diagnostic importance. Accordingly, last winter, it occurred to us that it might be of interest to gather together a series of cases of pleural effusion which we had examined with regard to the presence or absence of the paravertebral area of dulness. We fancied, at the outset, that on looking over the histories of the last two years we should find four or five cases in which there were satisfactory records of this phenomenon. Only one such case, however, was found. This instance and thirty-one others which we have observed during the past year, form the basis of the present communication.

Method of Procedure. Although percussion of the paravertebral triangle is a simple matter, it requires care and deliberation, as do all methods of precision. After percussing out the limits of the supposed effusion one should mark out the lower limit of pulmonary resonance on the opposite side and then percuss downward directly over the spine, marking the point at which dulness (relative) begins. It will be found usually that this point corresponds fairly closely with the beginning of relative dulness on the side of the effusion; that at all events it is a little higher than the beginning of flatness. One should then percuss downward along lines parallel to the spine and inward toward the spine along lines parallel to the lower limit of pulmonary resonance. In this manner it is usually easy to mark out, at the inferior and mesial angle of the healthy side of the back, a triangle of dulness. The vertical side of this right-angled triangle, represented by the line of the spinous processes, reaches to a point somewhat higher than the upper limit of flatness on the affected side. The base, represented by the mesial part of the line marking the lower limit of the lung on the unaffected side, varies, according to the size of the effusion, from 2 cm. to as much as 7 cm. in extent. The third side of this dull area is represented by a line joining the extremities of these two lines. It has sometimes seemed to us, as has been noted by Rauchfuss, that this line showed a slight outward convexity.

Summary of Observations. Of the thirty-two cases with which this report is concerned, twenty-four were instances of pleurisy with effusion. One of these was carcinomatous, and one was associated with a post-pneumonic infarction of the lung. Five were instances of hydrothorax in association with cardiac disease. Three were empyemas; one, post-pneumonic; one, typhoid; one, an interlobular, encapsulated empyema following puerperal infection. In all but one of these instances fluid was demonstrated by puncture or operation. In this case, an instance of infarction following pneumonia, there were signs suggestive of pleural effusion with a well-marked paravertebral triangle of dulness which disappeared later with improvement in the signs. No exploratory puncture was made. In the remaining thirty-one cases a distinct paravertebral triangle of dulness was demonstrated in twenty-nine.

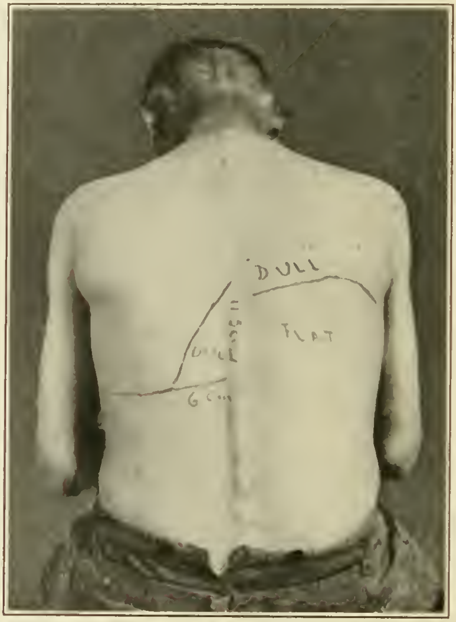

Fig. 4. Aortic insufficiency; hydrothorax on the right side; paravertebral triangle of dulness on the left.

Sex. Of these 29 cases, 19 were men; 10 were women.

The *age* varied from ten to sixty-seven years.

Side Affected. The effusion was on the right side in fifteen cases; on the left in thirteen, and on both sides, alternately, in one.

The *size* of the triangle varied with the extent of the fluid, the apex, when percussion was carefully carried out, reaching a point a little above the limit of flatness on the other side. The base line of the triangle ranged all the way from 3 to 7 cm. in length. The average size was slightly larger in right-sided than in left-sided effusions. In one case (No. 49553) a small, though distinct, triangle was made out in connection with a left-sided effusion, which on aspiration yielded only 250 c.c. of sero-fibrinous fluid. The upper limits of flatness of the effusion reached, in the back, to a point one finger's breadth below the angle of the scapula.

Variation in the Size of the Triangle with Change of Position. This important test was made in but four recent cases (Nos. 55951, 56297, 56479, a case in private practice). In each instance it was found that when the patient lay on the affected side the triangle nearly or entirely disappeared. In view of the fact that in the one case in which a suggestive paravertebral triangle of dulness was demonstrated in a pneumonia, change of position had little or no effect on the limits of the area of dulness, we are inclined to agree with Ewart in regarding this as one of the most important features of the sign.

No definite observations were made with regard to variations in the *vocal fremitus* over the paravertebral triangle. The area is indeed, so small and the subjacent muscle so thick that it is questionable whether variations in the fremitus could be easily brought out.

The character, however, of the *voice sounds* and *respiration* in Grocco's triangle are interesting. In 8 cases (Nos. 53903, 54097, 55278, 55218, 54731, 56297, 56762, a case in private practice) it was noted that the respiration and voice sounds were diminished in intensity over the area of dulness as compared with the adjacent parts. In five of these cases the respiration was of the same distant, tubular quality as that noted over the effusion, and in several instances the characteristic nasal character of the voice sounds was transmitted across into the area of triangular dulness. In the three cases in which it was tried, the coin sound heard over the triangle had the same clear, metallic ring as over the effusion. This metallic tinkling quality disappeared instantly when one passed out of the triangular area of dulness. In two instances in which a note was made as to the character of the respiration in the triangle, no change could be made out. In the remaining cases, no satisfactory note was made upon the auscultatory signs.

CASES OF PLEURAL EFFUSION WITHOUT THE PARAVERTEBRAL TRIANGLE. In two instances of pleural effusion, the paravertebral triangle was not observed. In the first one of these cases an instance of interlobar empyema, its absence, which was easily explainable, was of a certain diagnostic help. This case (53985–19392) was that of a woman, aged thirty-six years, who was admitted on March 3, 1906, complaining of pain in the left back which had come on during the puerperium, three weeks before. There was irregular fever and a marked leukocytosis. Everything pointed to some local suppuration. The local physical signs, dulness and suppressed respiration in the left back, were rather indefinite. On March 9 it was noted (Thayer) that flatness began in the left front at the seventh interspace in the anterior axillary line. There was deficient movement of the left chest; dulness throughout the back, amounting almost to flatness below the level of the angle of the scapula. In the dull area the vocal fremitus was almost, if not entirely, absent. On forced breathing there was a distant tubular quality. The voice sounds were distant and not nasal, except in a small area near the spine, where they sounded as if coming through an effusion. The cardiac apex impulse was not felt, but the heart sounds were heard best at a point 6.5 cm. to the left of the median line in the fifth space. Cardiac dulness (relative) extended 4 cm. to the right of the median line. Irregular fever and sweating continued. On March 26 a needle was introduced in several places in the back without result. On the same day it was noted that there was dull tympany at the left apex—a distinct Skodaic note; flatness at the eighth rib in the midaxillary line; deficient movement of the left back; percussion note, dull and high pitched, a little tympanitic as one passed down; not perfectly flat until one passed below the angle of the scapula. Vocal fremitus was felt through the back. *No paravertebral triangle of dulness at the right base.* The respiration in the upper part of the left chest in front was feeble but clear; a few fine rales on cough, increasing in number as one passed downward. Posteriorly, the respiration was enfeebled, of a somewhat tubular modification. Medium and fine moist rales below the spine of the scapular. Below the line of flatness respiration was much enfeebled, as were also the voice sounds; the same medium moist rales were heard following cough.

On April 9 it was noted that the left back was nearly flat; the dullest area lay between the posterior axillary line and a line between the scapular line and the spine. While vocal fremitus was felt along the spine and just inside of it, it was not so well felt a little farther out. The respiration was much enfeebled, distant, and tubular. The voice sounds were feeble over the duller area. *There was no paravertebral triangle of dulness on the right.* The history and physical signs, in connection with the absence of a paravertebral triangle of dulness, and the failure to find fluid on puncture, led to the diagnosis of either pulmonary abscess or interlobular empyema. On April 16 a needle introduced to a depth of 6 cm., just above the level of the angle of the scapula, revealed purulent fluid. Operation showed an interlobular empyema. The absence of a paravertebral triangle in this case is therefore easily explained; the fluid did not reach the spine.

The second case (No. 53475–19241) was that of a colored woman, aged thirty-one years, admitted to the hospital on January 16, 1906, complaining of abdominal pain and swelling, chills, fever, and sweating. The case was one of tuberculous polyorrhomenitis.

On January 18, 150 c.c. of hemorrhagic, somewhat cloudy fluid was removed from the right pleural cavity. On the following day flatness began at the fourth interspace in the right front, shifting with change of position, over two fingers' breadth. The limits of the dulness in the back were not noted. *No paravertebral triangle of dulness was demonstrable* although it was noted that there might "be a slight dulness in an area scarcely larger than 3 cm. square." Evidence of fluid gradually disappeared, although dulness with rather enfeebled respiration persisted at the right base, probably dependent upon pleural thickening. Examination in this case was made hastily and without great care. Percussion was carried out only to the left of the spine—*not over the spine.* It is probable that a more careful note would have revealed a more definite paravertebral triangle in the area referred to. The amount of fluid, moreover, was evidently small. No aspiration was, indeed, made after this second note, to prove positively the presence of fluid, although the movable dulness would appear to settle the question.

In a third instance the paravertebral triangle, which was demonstrated later, was not found before the first tapping. A. T. G. (No. 54210-19472), a colored waiter, aged twenty-six years, was admitted to the hospital March 21, 1906. There was a well-marked tuberculous pneumonia of the left chest, which, on percussion, was dull above and flat below. The respiration was tubular throughout, but feebler over the lower part of the chest. The vocal fremitus was not wholly absent at the base. *No paravertebral triangle was to be made out.* The heart was distinctly drawn toward the right, relative dulness extending 5.7 cm. to the right of the median line. On the following day, 800 c.c. of clear, straw-colored fluid was removed from the left chest by aspiration.

On April 12 it was noted that there was marked dulness throughout the left back, which was almost flat below the angle of the scapula; in this area the vocal fremitus was suppressed, the respiratory sounds distant, and the voice sounds nasal. *There was a well-marked paravertebral triangle* on the right, the base measuring 4 cm.

This is the only instance among our cases examined in which the paravertebral triangle of dulness was not demonstrated in the presence of a well-marked effusion. Its absence in connection with its subsequent appearance, we are unable to explain. Kernig, who found the sign constantly in a considerable number of cases, had one experience exactly similar to this.

Two Cases in Which a Paravertebral Triangle of Dulness was Detected in the Absence of a Demonstrable Effusion. In a number of cases of pneumonia and thickened pleura we have failed to detect a paravertebral triangle. Twice, however, the sign was observed in cases in which puncture of the chest failed to reveal fluid.

In the first instance (No. 54731–19642), in which, on entrance, there was a well-marked left-sided effusion with an associated paravertebral triangle of dulness on the opposite side, the base measuring 6 cm., aspiration of 1600 c.c. was followed on the succeeding day by an apparent reaccumulation. Two days later there was flatness to the angle of the scapula of the left side with a distinct paravertebral triangle measuring 9 x 6½ cm. Flatness in front began at the fifth rib in the recumbent posture, rising one interspace in the erect position. The breath sounds were clear in the upper front, feeble below; slight pleural rub in the third and fourth interspaces; voice sounds distant in the dull area. The left back was tympanitic above, flat at the angle of the scapula. The voice sounds were distant in the dull area, and the breathing feeble as one passed from above downward. There were a few crackles at the line of flatness. There was no egophony. Slight vocal fremitus was felt to the left base. The needle was introduced in two places, in the eighth interspace below the angle of the scapula, and in the ninth space. What was believed to be the lung was clearly felt, but no fluid was obtained. Two days later the signs were the same. Three days after this the patient insisted upon leaving the hospital. In view of the absence of fluid on puncture it is difficult to explain the persistence of the paravertebral triangle, unless one assumes that an existing accumulation was missed in the attempts at puncture—that which the sum total of the physical signs, especially the movable dulness in front, would seem to indicate. It may be that the needle was clogged each time with fibrin.

The second case was one in which the history and physical signs suggested pneumonia. The voice sounds in the affected area in the left back were, however, somewhat nasal and the respiration rather feeble. Percussion of the opposite side revealed a long but not very wide paravertebral triangle. The dulness was hardly as definite and marked as in our other cases. The apex, moreover, did not reach to the upper limit of the physical signs. *On change of position there was little or no change in the paravertebral area of dulness.* Exploratory puncture at two different points in the dull area in the left back was without result.

A small strip of dulness on the side opposite a massive pneumonia has been described by several observers. It is possible that the triangular shape may, in this instance, have been due to an accumulation of fibrin in the pleural sinus. The fact that there was no change in the limits of the dulness on change of position is worthy of note.

Notable Cases. Several cases seem to us to be deserving of special mention. There were three instances of encapsulated pleurisy, in two of which the paravertebral triangle was of real importance from a diagnostic standpoint.

The first one of these cases (No. 54097–19429) was that of a man, aged twenty-six years, who was admitted with pericarditis, with effusion following acute rheumatism. This was followed by pleuritic exudates, first on one side and then on the other. On February 21 it was noted that there was a well-marked area of flatness at the left base, especially in the lower interscapular region. Anteriorly to the scapular line, dulness was not marked. Over this area the vocal fremitus was diminished, almost absent; breath sounds distant, slightly metallic, and tubular; voice sounds nasal. The area of flatness as marked out during the visit may be seen upon the accompanying chart (Fig. 5). *On the opposite side there was a well-marked paravertebral triangle of dulness, the upper limit, 8.5 cm. from the base of the right lung, corresponding exactly to the area of flatness on the right; the base, 5 cm. in extent.* In the triangular area at the base of the right lung the voice sounds were nasal and the breathing tubular and feeble, of the same character as the sounds over the flat area in the left back. By aspiration, 400 c.c. of yellow fluid, with numerous leukocytes, was

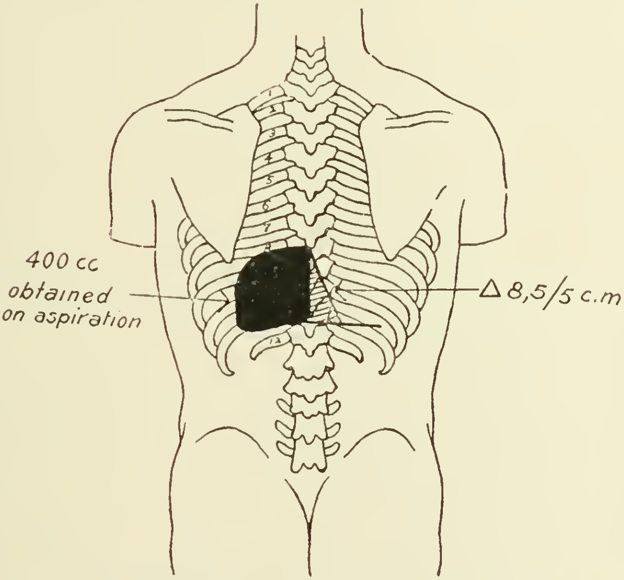

FIG. 5.—Paravertebral triangle in a case of encapsulated effusion. (Case 54097.)

obtained from the flat area on the left. Eleven days later, the resonance had cleared up on the left side, the flatness on the right side reached the tenth rib, while on the left a triangular area of dulness, 6 x 4½ cm., was to be made out. In a word, following the disappearance of the accumulation on the left side, there appeared a right-sided effusion with the development of a paravertebral triangle on the opposite side. Thus, in a single case, *the paravertebral triangle of dulness appeared successively on opposite sides of the chest.*

The second case (No. 54292) was that of a woman, aged forty-three years, admitted with acute lobar pneumonia. Before the resolution of the pneumonia, the temperature rose again, and on April 9 it was noted that there was dulness throughout the left back; the breath sounds were distant and feeble, tubular in the region of the left upper lobe and in the interscapular region. The voice sounds over the back were distinctly nasal. There was no absolute flatness in the left side of the chest. Just below and outside the angle of the scapula the voice sounds were almost egophonic. *On the right side of the chest a paravertebral triangle of dulness, measuring at its base about 3 cm., was clearly made out.* A needle, introduced in the lower part of the back, revealed pus, which was evacuated by the surgeon on the same day. Here an unresolved pneumonia complicated the picture and the presence of a paravertebral triangle of dulness of the opposite side was an important element in leading us to the diagnosis of effusion.

The third case (No. 54593) was that of a man, aged twenty-three years, admitted on April 22 with a severe typhoid fever. On May 10, with the patient lying on his left side, there was flatness below the angle of the right scapula with tympany above. The flatness ended sharply at the posterior axillary line. Over the flat area the vocal fremitus was diminished; respiration distant and tubular. There was a somewhat metallic quality to the respiratory murmur and the voice sounds were nasal. On the left *there was a paravertebral triangle of dulness measuring* 8 x 5½ *cm.* A needle introduced into the dull area on the right brought cloudy, purulent fluid. Operation revealed an encapsulated empyema.

In this case the demonstration of the paravertebral triangle was of material assistance in the diagnosis of an encapsulated empyema.

Conclusions. 1. In thirty-two cases of pleural effusion a paravertebral triangle of dulness at the base of the opposite chest (Grocco's sign) was clearly demonstrated in thirty. In one of the remaining cases, an instance with a small effusion in which the examination was rather hastily and imperfectly made, but a small, indefinite area of dulness was noted. In the other case the absence of the paravertebral triangle was easily explained by the position of the exudate—an interlobar empyema. In a third instance, in which a triangle was demonstrated later, it was missed on the first examination. The sign, therefore, may be regarded as practically constant in cases in which there is free fluid in the pleural cavity, or in which an encapsulated effusion impinges on the spine.

2. This triangle is an area of relative dulness, the vertical side of which is represented by the line of the apophyses of the vertebræ, the apex reaching a point somewhat above the level of flatness of the effusion; the base is formed by the lower limit of pulmonary resonance on the healthy side for a distance of from 2 to 7 cm. from the spine; the hypothenuse, by a line connecting these points. This last line shows, sometimes, a slight outward convexity. The dulness is more marked as one approaches the spine.

3. In the triangular area of dulness the respiratory murmur is often suppressed and of a quality similar to that heard over the effusion. Egophany or a nasal quality of the voice sounds may also be present. The coin sound, when the manœuvre is carried out by placing the coin on the front of the chest below the level of the effusion, shows the same metallic ring as that which is observed over the affected side. These signs may be so marked as to make it possible to outline the area of the triangle by auscultation.

4. When the patient lies on the affected side the triangle of dulness diminishes or disappears, and returns again when the erect posture is assumed, or when the patient lies on the other side.

5. The triangle is usually larger on the left side—that is, in the case of right-sided effusions—than on the right.

6. In three cases of encapsulated effusion the presence of a paravertebral triangle of dulness on the opposite side proved of real diagnostic assistance.

7. Our experience justifies the conclusion that the paravertebral triangle of dulness is a remarkably constant and characteristic sign of fluid in the pleural cavity. It further supports the assertion of the discoverer that it may be of particular value in the case of encapsulated effusions. We regard the sign as an important addition to our means of diagnosis of effusions in the pleural cavity.

8. As to its cause, we are inclined to accept the explanation offered by Baduel and Siciliano, namely, that the fluid, lying against and passing anteriorly over the bodies of the vertebræ, acts as a mute in suppressing the sonorous vibrations of the spine. This deadening of the resonance is naturally appreciable for a certain distance beyond the median line over the area occupied by the transverse processes and the first part of the ribs. It is more marked and extends over a wider area at the base, where the fluid, collecting in the sinus of the pleura, comes into wider contact with the vertebræ and pushes farther around toward the opposite side. The displacement of the contents of the mediastinum—aorta, œsophagus, azygos vein, heart—may play a part, especially in effusion on the right side of the chest, in producing this dulness as well as in bringing about a certain degree of compression of the lung on the opposite side.

9. Although there may be a small strip of dulness along the spine on the side opposite a pneumonia, we have but once detected anything approaching a paravertebral triangle. In this instance the dulness was not very definite and showed little or no change on change of position.

ADAMS-STOKES DISEASE (HEART-BLOCK) DUE TO A GUMMA IN THE INTERVENTRICULAR SEPTUM.

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The condition at present recognized as the Adams-Stokes syndrome was first described by Adams[1] in 1827, and more fully studied by Stokes[2] in 1846. Since that time many cases have been reported and studied, but until very recently no really convincing demonstration of the etiology and pathological basis of the condition had appeared. Thus, Charcot held that the lesion was to be found in the medulla; Stokes himself suggested the heart muscle; Huchard[3] held that the circulatory mechanism of the heart, together with disease of the cerebral vessels, should be held accountable; Gibson[4] and Jacquet[5] called attention to a certain similarity with intermittent claudication; and others have ascribed the condition to fatty degeneration of the heart, to tumors of the vagus, or to pressure upon the pneumogastric nerve. Gaskell[6] was the first to demonstrate the fact that, in tortoises, constriction of the circular layer of muscle at the auriculoventricular junction inhibits the normal rhythm of the heart, so that the auricles and ventricles beat independently of each other. Chauveau,[7] Vaquez and Bureau,[8] and Moritz[9] have contributed interesting articles dealing with the dissociation of the auricles and the ventricles.

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[1] Dublin Hosp. Reports, 1827, vol. iv, 396.

[2] Dublin Quart. Jour. Med. Sci., 1846, ii, 73.

[3] Traité du Maladie du Coeur.

[4] The Nervous Affections of the Heart, 1904, p. 65.

[5] Deut. Arch. f. klin. Med., lxxii, No. 2.

[6] Jour. Physiol., 1883, iv, 43.

To Erlanger[10] belongs the credit of first suggesting that the lesion of Adams-Stokes disease lay in the bundle of His, and of offering incontestable experimental evidence supporting his belief. In 1905 Stengel[11] made a preliminary report of a case of Adams-Stokes disease in which the autopsy disclosed macroscopically an atheromatous lesion on "the anterior mitral leaflet toward its base and aortic edge," being, therefore, in the region "exactly over the bundle of His where this band passes from the ventricle to the auricle." Thus far there has been no report of the microscopic findings, so that it is still uncertain whether there was actual involvement of the bundle, although there is strong presumptive evidence that such was the case.

For many years an active discussion has been carried on by physiologists as to the cause of the rhythmic activity of the heart. One school—the followers of the myogenic theory—has held that the heart muscle itself possesses an inherent automaticity, which is influenced by, but not dependent upon, the nervous mechanism. The other school took the opposite view, namely, that the nerves and ganglia are of primary importance. Long ago the fact was pointed out that contractions occur in the fœtal heart before its nervous structures are developed, and this has tended to support the myogenic theory.[12]

It has been demonstrated by the experiments of Gaskell, His, Tigerstedt, and Erlanger that the symptoms of Adams-Stokes disease and those of heart-block, brought about by injury to the bundle of His, are very similar. Erlanger, by an ingenious clamp screw applied to the muscular bundle, was able to bring about a gradual failure of conductivity from auricle to ventricle. When slight pressure was made the ventricle would miss every fourth auricular stimulus; when the pressure was further increased, the ratio would fall from 1 to 3 to 1 to 2, and finally complete dissociation of the rhythm would occur. In other words, a condition of complete heart-block had occurred, and the ventricle setting up a rhythm of its own contracted entirely independently of the auricle. "With each change in rhythm the blood pressure was much affected, falling with the fall of the ventricular rate and rising with its increase. But even in complete block a fair pressure might be maintained. With partial block both auricle and ventricle might be inhibited by stimulation of the vagus as easily as under normal conditions. But when the block was complete no diminution, or only a slight diminution, of the rate of the ventricle resulted from stimulation of the vagus. The auricles reacted normally to vagus stimulation. Section of both vagi had little or no influence on the rate of the ventricular beat when the block was complete. But stimulation of the accelerator nerve usually increased the rate of the ventricle. The reaction of the auricles to stimulation of the accelerator nerve was not influenced by the block."

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[7] Rev. de Med., 1885, v, p. 161.

[8] Compt. rend. du Soc. Biol., Paris, 1893, p. 170.

[9] St. Petersburg med. Woch., 1897, p. 301.

[10] Jour. Exper. Med., November 25, 1905; Ibid., January 25, 1906, p. 8; Bull. Johns Hopkins Hosp., June, 1905.

[11] Amer. Jour. Med. Sci., 1905, cxxx, 1083.

[12] Erlanger, Jour. Amer. Med. Assoc., 1906, June 2, 1895.

In mammalia the muscular structure of the auricle is separated from that of the ventricle by a layer of connective tissue. In order to substantiate the contention of the upholders of the myogenic theory of cardiac rhythmicity it was essential that some bridge of connecting muscular tissue be demonstrated. This was accomplished by Wm. His, Jr.,[13] and by Stanley[14] independently of each other. But His was the first to succeed in sectioning this muscular bundle, and bring about a dissociation of the auricular and ventricular rhythm.

Owing to the meager character of His' preliminary publications which appeared in 1893, the subject attracted little attention until 1904, when the existence of the bundle, its physiological properties, and its exact anatomy were confirmed through the researches of Retzer,[15] Breunig,[16] Humblet,[17] and Hering.[18]

The structure which is now known as the bundle of His consists of a narrow band of fibers 18 mm. in length, 2.5 mm. in width, and 1.5 mm. in thickness, extending from the right auricle and its valves to the interventricular septum.

More recently the structure has been carefully studied by Keith[19] and Tawara.[20] The latter has given a developmental as well as an anatomical description of it. According to him the "bundle of fibers passes forward from the coronary sinus through the interauricular septum to the central fibrous body of the heart, on which it forms a plexus. One of the bundles from this plexus breaks through the fibrous body into the interventricular septum and is distributed subendocardially on the right and the left sides of the latter. The auricular part of the bundle is made up of very fine, richly nucleated fibers, and the fibers of the ventricular part belong to the Purkinje type."[21]

Still further corroboration of the auriculoventricular dissociation which occurs in Adams-Stokes disease has been furnished by Gibson,[22] who has studied the electromotive changes in a case of heart-block by means of Waller's apparatus. By this method the electric variations are indicated by means of a Lippmann capillary electrometer. "Leading off from the basal region of the precordium to the acid, and from the apical region to the mercury, the usual diphasic movements of he capillary column were clearly seen preceding the apex-beat, and evidently resulting from the ventricular systole. But in the interval between these waves other smaller waves were distinctly seen, and can be attributed only to the systole of the auricle."

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[13] Arbeit. a. d. Klin. zu Leipzig, 1893, p. 23; Zentrbl. f. Phys., 1895, ix, 469; Deut. Arch. f. klin. Med., 1899, lxiv, 316.

[14] Jour. of Physiol., vol. xiv.

[15] Arch. f. Anat., 1904, p. 1.

[16] Arch. f. Physiol., 1904, suppl. 1.

[17] Arch. internat. d. Physiol., 1904, i, p. 278.

[18] Pflüger's Archiv., cviii, p. 267.

[19] Lancet, 1906, i, 623.

[20] Vide Aschoff, Münch. med. Woch., September 26, 1905; Centrbl. f. Physiology, 1905, xix, 70, 298.

[21] Ewart, Progressive Medicine, September, 1906.

The electromotive changes have also been most carefully studied by Einthoven,[23] on a still more elaborate scale. Curiously enough the recording instrument was placed in the physiological laboratory while the patient remained in the hospital. The pulsations were transmitted by means of wires. "He employed a specially arranged galvanometer, the movements of which were recorded on a travelling photographic plate, on which two ordinates and abscissæ, representing electric potential and fractions of a second respectively, were simultaneously recorded." In making tracings for a case of heart-block he was able to demonstrate complete dissociation of the auricular and ventricular oscillations. This method, while obviously impractical in a clinical study of cases, owing to the fact that in addition to a complicated apparatus trained observers with a special knowledge of physics, are necessitated, nevertheless presents very accurate and interesting corroboration of other recent investigation.

SYMPTOMS. The cardinal symptoms of Adams-Stokes disease consist of (1) bradycardia, (2) cerebral attacks, and (3) pulsation of the cervical veins, exceeding in rate the arterial pulsation.

The Pulse. In most cases the pulse is habitually slow, sometimes reaching 30 or 20 beats per minute. Often the rate bears a definite relation to that which was normal for the individual in question. Thus, it may be one-half or one-quarter as fast. This is due to the fact that only every other or every second stimulus is able to cross the bundle of His. The arterial pulse is generally regular in rhythm, although arrhythmia is not uncommon. During the acute attacks the ventricular contractions may fall as low as 10, 8, or even 3 beats per minute. Kidd[24] and Laslett[25] found a condition of asystole lasting sixty and ninety seconds respectively. In Stengel's case an interval of two minutes and ten seconds was noted. If the heart is auscultated during these periods one may hear feeble and muffled contractions. These have given rise on the part of some authors to the belief that the sounds are due to abortive systoles, too weak to make themselves felt at the wrist. The most generally accepted opinion, however, is that they are due to contraction of the auricles, a condition which, as Gibson and Mallet[26] have long since shown, does produce a definite sound. When simultaneous tracings of the arterial and the venous pulses have been made it has nearly always been possible to prove definitely the etiological agency of the auricle. At times it is also possible to *see* that the jugular pulsation in the neck is synchronous with the doubtful sounds. Maynard's[27] contention, based upon two cases with radial tracings only, that the accessory heart sounds were due to hemisystole, and not to the contraction of the auricles, is not convincing.

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[22] Brit. Med. Jour., 1906, ii, 22.

[23] Arch. neerl. d. sc. exactes, Harlem, 1906, ser. 2, tome xi.

[24] Lancet, February 13, 1904.

[25] Lancet, June 4, 1904.

The slow pulse in Adams-Stokes disease is due to depression of conductivity in the auriculoventricular bundle. The auricles contract more or less normally, but the ventricle only responds to every other, every second, or every third stimulus. Just upon what the exacerbations of bradycardia depend still is uncertain.

Norfleet[28] has reported a case of Adams-Stokes disease occurring in a man of sixty-nine years, who for over a year had a pulse-rate of 45 or less per minute. With exacerbations of the bradycardia there were convulsive movements. During these attacks the pulse-rate sometimes remained as low as 8 or 9 per minute for periods of twenty-four hours.

The cerebral symptoms consist of attacks of weakness, vertigo, unconsciousness, occasionally convulsive seizures, and at times Cheyne-Stokes respiration. The patient may have an uneasy feeling about the heart, becomes pale and promptly falls unconscious, being practically pulseless at the wrist, and soon breaks out in a clammy sweat, consciousness returning gradually. The attacks may last from a few seconds to several minutes, they may be repeated at varying intervals and anyone of them may end fatally. Upon returning to consciousness the patient often states that he had a nightmare—thought he was dying.

Occasionally attacks like those just described alternate with simple vertigo, but some cases present certain features which closely resemble apoplexy, the patient falling rapidly into a deep coma, with flushed face and stertorous breathing. In one of Osler's[29] cases there was transient motor aphasia. Actual convulsions, as happened in our case, may also occur. When present they are mainly limited to the face and hands, although general epileptiform attacks are not unknown.

In some cases the initial pallor of the face may be replaced by congestion. In our case this was a very noticeable feature. A sensation of blood rushing to the head was a marked symptom in Leuchtweis' case. The convulsive attacks occurring in Adams-Stokes disease are probably the result of cerebral anemia. They are apparently similar to those produced experimentally in animals by severe hemorrhage. The apoplectiform symptoms may reasonably be assumed to be due to venous congestion of the brain, a condition which has long been known clinically to be associated with bradypnœa, paresis of the glottis, and spasmodic conditions.

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[26] Jour. Anat. and Physiol., 1879, xiv, 4.

[27] Brit. Med. Jour., 1905, ii, 847.

[28] Medical Record, November 21, 1903.

[29] Lancet, 1903, ii, 516.

The belief held by Tripier[30] that the convulsive attacks preceded and were the cause of the bradycardia has been definitely disproved by Webster's[31] tracings.

Some cases exhibited features common to other cardiac disorders, such as nocturnal asthma, precordial oppression, pallor, sudation, and dyspnœa.

The case that we report herewith is of very considerable interest: It presented all the cardinal symptoms of Adams-Stokes disease; sphygmographic tracings taken coincidently from the jugular vein, the radial pulse, and the apex beat showed the incoordination of the auricles and ventricles, the former contracting more often than the latter; and at autopsy a growth was found involving the auriculoventricular bundle, which on microscopic examination proved to be a gumma.

According to His, Jr.,[32] dissociation of the auricles and ventricles might be brought about by any one of three conditions: (1) Degeneration of the myocardium of sufficient extent to interfere with conductivity; (2) dromotropic nerve influences—inhibitory impulses—conveyed principally through the agency of the pneumogastric; and (3) localized disease of the auriculoventricular bundle. As the microscopic examination shows, our case belonged to the last named category.

E. B., a white, male, aged thirty years, a native of Ireland, was admitted to the Philadelphia General Hospital May 20, 1906, complaining of vertigo and syncopal attacks.

Family History. Father died from accident; mother from senility, aged ninety years. Mother had seven children; with the exception of one who died of pertussis at four years, all are living and well.

Personal History. Previous illnesses: Pertussis, mumps, scarlatina, and diphtheria in childhood. Typhoid fever in 1895, lasting two months (including a relapse). Ever since then he has had a trifling but persistent cough. The patient was brought up in the country until he was fourteen years of age, at which time he secured employment in a hardware store. At eighteen years he enlisted for three years in the British Army. Afterward he spent eleven years frequently changing his occupation, having for a time been a barkeeper. He then again enlisted, serving in Cairo, India, and seeing actual service in the Boer war. During the last two years he has been a waiter. He has indulged in occasional alcoholic intoxication, but this was exceptional; he has used tobacco to excess. In 1893, he contracted a "chancroid" of the penis, which appeared eight days after intercourse, and lasted eleven days. He received only local treatment. The sores were multiple, he never developed any rash or constitutional symptoms. He has been married two years. His wife has borne two healthy children, and has never had any miscarriages. He has never had any gastrointestinal, or renal symptoms.

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[30] Rev. d. Med., 1883, p. 1001.

[31] Glasgow Hosp. Reps., 1901, p. 413.

[32] Deut. Arch. f. klin. Med., lxiv, p. 316.

Present illness began on May 16, four days before admission, when while riding in an electric car asleep, he had an attack of syncope. He woke to consciousness to find the other passengers looking over him. During the attack he had fallen from his seat. He describes the onset of the attack as beginning with the sensation that blood was rushing to his head, associated with some vertigo which increased. This phase of the attack was very brief, and he was unable to state whether objects seemed to whirl about him, or he about them. He had two similar attacks subsequently on the same day, and on the following day two or three more. He had three attacks on the 18th, and on the 19th had an attack about 1 A.M., while in bed, after a hard day's work. On the day of admission he had attacks every ten or twenty minutes throughout the day.

Physical Examination. The patient is well-developed and nourished. The pupils are equal, and react promptly to light and distance. The ocular movements are normal; the tongue is fairly clean. The chest is well-formed; the expansion equal and of normal extent. The lungs are resonant and clear throughout. The extremities are negative except for decided downward curvation of the finger-nails, and a scar on the right knee, resulting from a former bullet wound.

In the *recumbent posture* the cardiac dulness extends from the right sternal border, and the upper border of the third rib, to the midclavicular line. The apex-beat is fairly diffuse, and is visible and palpable in the fifth interspace. The radial pulses are equal and synchronous. The blood pressure is low—systolic 117 mm. Hg. (Stanton), diastolic 85 mm. There is marked pulsation of the veins of the neck, the rate being about three times as rapid as that of the radial pulse. This pulsation is direct (not transmitted from the carotid) and is slightly arrhythmic. The first sound at the apex is feeble, and it is followed after a fraction of a second by a second sound, apparently muscular in character, which can be clearly differentiated from the normal second sound of the heart, which follows it, and may be heard over the entire precordium. There is a systolic murmur at the apex, which is transmitted toward the axilla, but not to the angle of the scapula. The ventricular beats are all felt at the wrist. The pulse rate is 48.

In the *sitting posture* the heart sounds become louder, the systolic murmur is more intense, and the irregularity more marked. The radial pulse-rate is unaffected by the change in posture, still remaining 48. The liver can be palpated at the level of the umbilicus.

May 24. The patient has had three attacks since admission, until today, when they have become more frequent, occurring about once every half to three-quarters of an hour. The attacks begin with a sense of fulness of the head, vertigo, and end in syncope; and last altogether about five seconds. In one attack he had twitching of the facial muscles.

May 23. *Ocular examination*, by Dr. Hansell. There is a most unusual condition of the vessels of the right disk. The artery superficial to the vein is distended in an angular shape, and communicates with the large underlying vein by a broad channel—arteriovenous aneurysm. The arteries beyond the disk contain venous blood. The left fundus is healthy, but darker than normal, owing to the number and tortuosity of the veins.

May 28. *Nasal examination* by Dr. Roberts discloses a deviation of the septum toward the left, both bony and cartilaginous. Except for pallor of the mucous membrane there is no other abnormality. The pharyngeal mucous membrane is somewhat glazed and slightly congested.

The larynx and vocal cords are normal.

Urine. 1020, alkaline, no albumin, no glucose; triple phosphates and amorphous urates.

Blood. Hemoglobin, 80 per cent.; erythrocytes, 3,136,000; leukocytes, 7800; polymorphonuclear cells, 77 per cent.; lymphocytes, 23 per cent.

May 25. *Nurse's Report.* During the attacks the patient becomes weak; objects seem to move around; failure of eyesight for two or three minutes; the face becomes red, and the breathing increases in rapidity for a few seconds. He has had two such attacks within an hour. They usually come on after some unusual occurrence in the ward.

3.30 P.M. Another attack; feels flash of heat over body; head is light; eyes heavy; the radial pulse drops several beats. 4.30 P.M. Slight convulsive spell; twitching of hands—head thrown back—duration one minute.

June 3. Patient is out of bed and about the ward. Complains of weakness. No attack for five days.

June 7. Two attacks yesterday. During attacks pulse feeble and irregular. On auscultation heart sounds are fainter. The respiratory sounds increase in depth, rate and intensity. Face flushed—twitching of hands. Some attacks are preceded by rolling of the eyes, which is followed by a tremor of the body and hands, the latter quite forcibly at the onset. Attacks sometimes last only from seven to ten seconds.

June 8. Has had a series of attacks. Extremities cold, complains of it; cyanosis; pulse-rate generally about eighteen per minute, increasing at frequent intervals to thirty. After one such acceleration the heart stopped for fifteen seconds. Twitching hands and staring eyes. Patient considered the last a slight attack. Has had frequent attacks during the last twenty-four hours. Vomits a good deal, is very weak.

June 9. Has vomited everything put in his stomach since yesterday. Attacks numerous; asthenia marked. Toward evening he became able to retain peptonized milk, and finally had a restful night without seizures.

June 10. Continued improvement; no more attacks; stomach retentive; appears stronger. At night an attack seemingly like the rest proved fatal. The patient a few minutes before death had been resting quietly.

During his stay in the hospital the patient received in addition to certain drugs administered for their effect upon the gastrointestinal tract, strychnine, nitroglycerin, and sodium iodide. None of these had any effect upon the cardiac condition. The pulse-rate remained unchanged; the attacks were neither retarded nor abated. It is hardly likely that the iodide would have been beneficial even if the patient had lived longer, for as the subsequent microscopic examination of His' bundle showed, there was very great destruction of tissue.

The accompanying tracings (Figs. 1, 2, and 3), taken a few days after the patient's admission, show very distinctly that the bradycardia was due to a disturbance of conductivity. The tracing from the jugular vein proves not only a diminution in the function of conductivity as evidenced by the lengthening of the A-C interval—the length of time required for the stimulus to pass from the right auricle to the left ventricle, but also the fact that many of the auricular contractions evoked no response whatever on the part of the ventricle. In other words, we have to deal with a condition of complete "heart-block," or what is a better term, auriculoventricular dissociation. It is also to be noted that the frequency of the auricular contractions was markedly increased by the forced inspiration which occurred during the later part of the tracing, whereas the ventricular wave underwent no corresponding change. This is, of course, further evidence of disturbed conductivity. In the cases reported by Stengel, Dock,[33] and Finkelburg,[34] the administration of atropine had no effect whatever upon the rate of the ventricular contractions. This is due to the fact that atropine in man, as in animals, exerts its influence upon the heart through depression of the pneumogastric, which affects the auricles directly, whereas the ventricles, if the contraction stimulus cannot pass through the bundle of His, are uninfluenced by it. In Schmoll's[35] case the administration of atropine reduced the auricular cycle from twelve-tenths to eight and one-half tenths of a second, while the ventricular time underwent no change.

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[33] Med. News, Aug. 19, 1905.

[34] Deut. Arch. f. klin. Med., April 6, 1905.

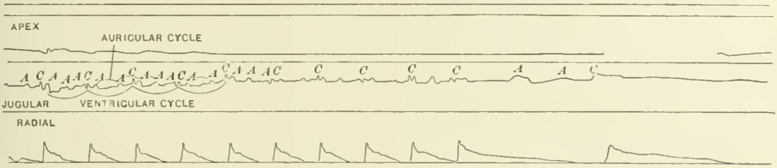

FIG. 1

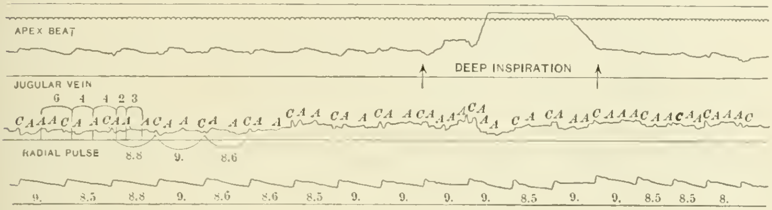
APEX BEAT
DEEP INSPIRATION
JUGULAR VEIN
6 4 4 2 3
C A A A C A A C A A A C A A C A A C A A C A A C A A C A A C A A A C A A A C A A C A A A C A A A C A A C A A C A A A C A A C A A A C
RADIAL PULSE
8.8
9.
8.6
9.
8.5
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FIG. 2

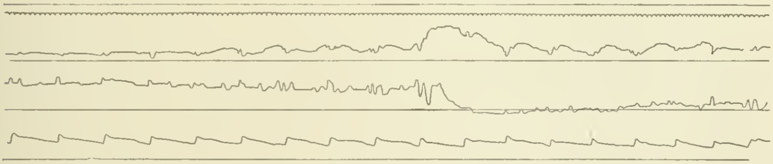

FIG. 3

Cardiosphygmographic tracings. In Fig. 1, during the last two ventricular contractions the speed of the cardiosphygmograph was increased about fourfold. The duration of the auricular cycle varied from four-tenths to twelve-tenths of a second, that of the ventricular cycle from seventeen-tenths to eighteen-tenths of a second. *A*, auricular systole; *C*, ventricular systole. The time marked records one-fifth second.

The fact that the ventricles were not affected by vagus influence was shown by the fact that change of posture on the part of the patient—from the recumbent to the sitting, and from the latter to the standing position—had no influence whatever upon the frequency of the radial pulse. A further study of the tracing shows that the failure of the ventricle to respond to the auricular contraction was not due to any loss of power on the part of the ventricle. The elevations in the systolic phase of the radial tracings are large, powerful, regular in strength, and the blood-pressure is normally sustained. This has also been substantiated by the microscopic examination of the ventricular muscle. The sphygmogram also demonstrates the fact that the accessory heart sounds heard over the precordium between the radial pulses must have been auricular in origin, and were not due to abortive ventricular systoles, since the radial pulse follows regularly each ventricular contraction, as is shown by the apical tracing.

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[35] Jour. Amer. Med. Assoc., 1906, xlvi. 361.

It will be noted that the ventricular cycle required about nine-fifths of a second; the auricular cycle only from three-fifths to six-fifths. The tracing also shows that there is no definite relation between the auricular and the ventricular contractions. If we were simply dealing with a condition of depressed conductivity we should find the A–C interval either constant in time or increasing in length. This is not the case; the A–C interval at the beginning of the tracing steadily decreases in time until the auricle overtakes the ventricle—indicating complete block. In the latter half of the sphygmogram the deep forced inspiration—between the pointers—produced a marked acceleration of the auricular contractions, while the ventricular rate underwent practically no change.

Additional evidence as to the accuracy of the interpretations of the various phases of the jugular pulse in cases of Adams-Stokes disease has been furnished by a number of observers. Ritchie,[36] Gibson, Finny,[37] Villaret, and Brouardel[38] examined their cases by means of the fluoroscope and were able to demonstrate auriculoventricular inco-ordination.

Autopsy. The body is that of a moderately well-nourished adult male; weight, approximately 154 pounds. Rigor mortis is marked; cadaveric lividity slight. Panniculus adiposus is moderate. The muscles are of good volume, bright red in color.

The peritoneum contains a small amount of translucent straw-colored fluid. The abdominal organs are normal in position and relations. The spleen weighs 320 grams, and measures 15 x 10 x 6 cm. The consistency is fairly firm; the cut surface dark red in color, and bulges slightly; the follicles are prominent and well-defined; the trabeculæ are moderately prominent. The *liver* weighs 2050 grams; and measures 28 x 23 cm. The surface is smooth, dark reddish-brown in color with lighter mottlings. It cuts with slightly increased resistance; the cut surface shows normal liver markings. In the posterior portion of the right lobe is a spherical nodule 1 cm. in diameter, dark bluish-gray in color, of a spongy consistency. The *pancreas* presents no pathological features. The *stomach and intestines* aside from vascular injection present no unusual features. The *suprarenals* are somewhat smaller than normal; the cortex brownish-yellow. The *left kidney* weighs 190 grams, is firmer than normal, cuts with ordinary resistance, its surface does not bulge, and the capsule strips readily. The cortex and medulla preserve their normal relation in width and are well differentiated. Pyramids are dark reddish-purple and streaked, the cortex considerably lighter, showing normal markings. The *right kidney* weighs 250 grams and presents the same characteristics as the left. The *ureters* show no abnormalities. The *bladder* contains a small amount of turbid, yellowish urine. The walls are of normal thickness; mucous membrane deeply injected. The *left pleural cavity* contains 300 c.c. of translucent straw-colored fluid. At the base are a few firm, fibrous adhesions. The *right pleural cavity* contains 100 c.c. of fluid of the same character as the left. The opposed surfaces of both pleural cavities, not involved in adhesions, are smooth and glistening. The *left lung* weighs 690 grams, and crepitates throughout. On section the lung is light pinkish-gray in color. On pressure a considerable amount of slightly blood-tinged, frothy fluid is exuded. The *right lung* weighs 490 grams and presents the same features as the left. The *pericardium* contains about 20 c.c. of translucent straw-colored fluid. The opposed surfaces are smooth and glistening.

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[36] Proc. Royal Soc. Edin., 1905, xxv, 1085.

[37] Brit. Med. Jour., 1906, ii, 967.

[38] Arch. d. Méd. Exper., 1906, xviii, 230.

The *heart* weighs 510 grams. Both ventricles and the right auricle contain a small amount of cruor and chicken-fat clot lightly adherent to the wall. The wall is somewhat thinner than normal. The right auriculoventricular orifice admits four fingers with ease. The tricuspid leaflets present no pathological features. A small amount of cruor clot is adherent to the wall of the right ventricle. The right ventricular wall measures 4 mm. in thickness. The papillary muscles are of fair size. The cavity of the left auricle is apparently not enlarged. The wall is of normal thickness. The left auriculoventricular orifice admits two fingers with comparative ease. The mitral leaflets show some fibrous nodular thickening along the line of closure. The leaflets are not distorted. The endocardium and myocardium of the left ventricle present, instead of their normal appearance, the following characteristics: excepting in the posterior portion, from the insertion of the mitral leaflets to the apex, there is a mass protruding approximately 4 mm. above the surface of the normal endocardium, considerably firmer in consistency than the cardiac muscle. The involvement of the left ventricle in this mass can be seen in Fig. 4, the area through which the incision (A–B) runs being the portion of the interventricular septum especially affected, and C, D, and E the papillary muscles through which the process has extended. The endocardium in this region is opaque, grayish-yellow, the surface somewhat nodular. As the middle portion (A, Fig. 4) of the interventricular septum is incised, is seen a firm mass, extending from the endocardium 1.5 cm. toward the right ventricle, and containing a number of homogeneous, yellowish, structureless areas, varying in diameter from

0.5 to 2 cm., and surrounding them areas of a grayish translucent appearance. As the upper portion (B, Fig. 4) of the interventricular septum is incised, this mass is seen to involve practically the entire thickness of the septum, presenting on the right ventricular surface of the septum in three nodules, the one (A, Fig. 5) measuring 1.5 cm. by 1 cm., at the base of the papillary muscle of the middle tricuspid leaflet; the second (B, Fig. 5), somewhat larger, a little above this; and the third (C, Fig. 5) at the base of the posterior tricuspid leaflet.

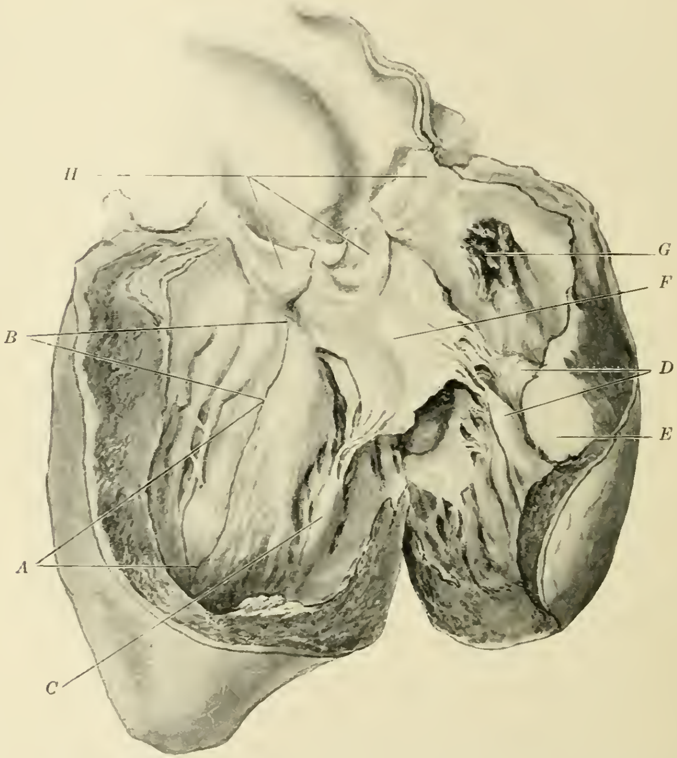

Fig. 4.—The cavity of the left ventricle, showing the gummatous involvement of the wall. *A*, *B*, incision into the gummatous mass, involving the interventricular septum; *C*, *D*, *E*, papillary muscles to which the process has extended; *F*, anterior mitral leaflet; *G*, small clot adherent to the ventricular wall; *H*, aortic leaflets.

All of these nodules consist of a central, firm, homogeneous, yellowish mass, surrounded by dense, grayish, rather translucent tissue. Nowhere within this mass can any tissue be seen of the macroscopic appearance of normal cardiac muscle. At the borders, the demarcation of this mass from the cardiac muscle is comparatively sharp. Upon the posterior superior portion of the endocardium of the left auricle is a thin grayish-red clot, measuring 2 by 1.5 cm., firmly adherent to the endocardium in its central portion. About the

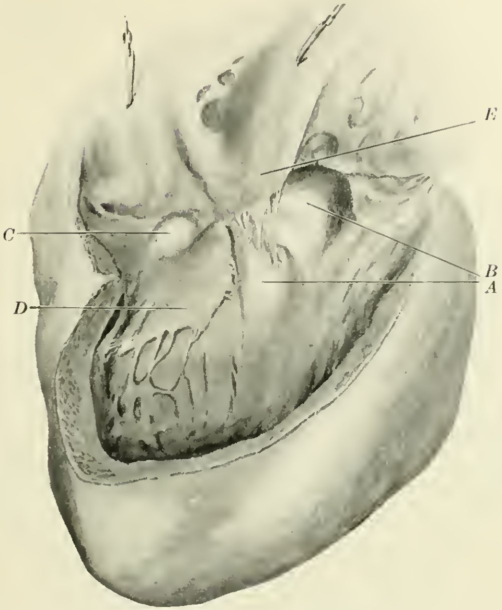

Fig. 5.—The cavity of the right ventricle, showing the extension of the gummatous infiltration from the left ventricle completely through the interventricular septum. *A*, *B*, *C*, gumma presenting on the right ventricular surface of the interventricular septum; *D*, posterior tricuspid leaflet; *E*, middle tricuspid leaflet.

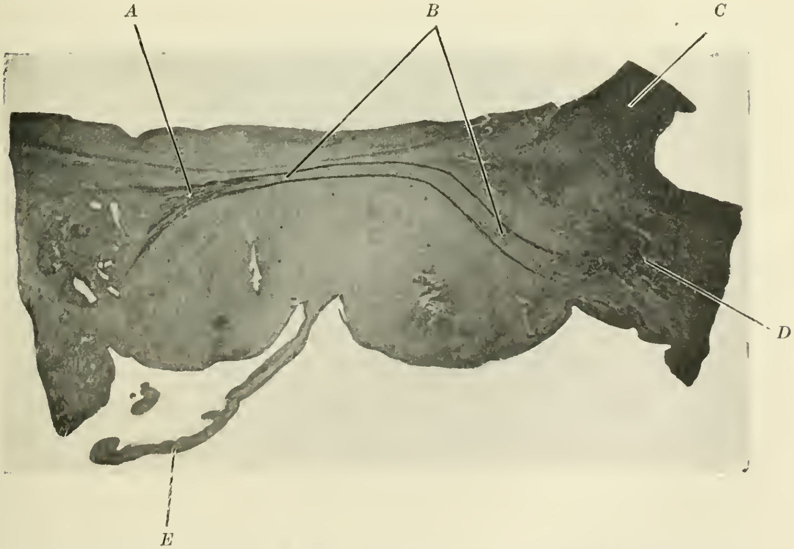

Fig. 6.—Photograph (slightly enlarged) of Section No. 1, with a tracing, showing the course of the bundle of His, from the interauricular septum to its bifurcation. *A*, the bifurcation of the bundle of His; *B*, the course of the bundle; *C*, base of the anterior mitral leaflet; *D*, the interauricular septum; *E*, middle tricuspid leaflet.

middle of the endocardial surface of the left ventricular portion of the interventricular septum is a similar area measuring 0.3 by 0.6 cm. The endocardium under these clots is rough and granular. The left ventricular wall, free from involvement in the mass above described, is 1.2 cm. in thickness. The portions involved in this mass vary from 1.4 cm. to 1.8 cm. in thickness. There are no signs of sclerosis in either coronary arteries or any of their branches as far as they can be followed. Slightly above the aortic leaflets are a few small yellowish areas, pin-head in size. There is no sclerosis and no plaque formation.

The only features of interest in the histological examination of the tissues, excepting those of the heart, are a moderate degree of chronic passive congestion of the spleen, liver, kidneys, pancreas, intestines, stomach, and lung; slight œdema of the lungs; and moderate atrophy of the cortical tissues of the adrenals.

Histological examination of the heart (not including the auriculoventricular bundle of His): The cardiac muscle not involved in the growth above described shows only a moderate amount of fragmentation with slightly decreased prominence of the transverse striations. The portions particularly involved in the pathological process present somewhat varying pictures. In those areas where the process is most advanced there is found centrally a necrotic, homogeneous mass, staining moderately well with eosin, and containing, especially toward the periphery, some nuclear dust. Nowhere within this area is any cell structure preserved. Surrounding this necrotic area is a zone composed primarily of lymphocytes with some polymorphonuclear leukocytes and plasma cells, and a considerable number of young bloodvessels with walls composed of a single layer of endothelium. Toward the periphery numbers of small spindle-shaped cells appear and some larger epithelioid cells, while lymphocytes and plasma cells are still present in small numbers. Young bloodvessels become much more numerous. In a number of areas outside of this zone can be seen one composed of fibrous connective tissue, some of which have undergone hyaline degeneration. In none of these areas are giant cells to be seen. The cardiac muscle immediately surrounding these areas is, in most cases, extensively disintegrated and here and there can be seen a few fibers apparently undergoing hyaline degeneration. The areas in which the process has not proceeded to the stage above described present two pictures. In some of them there is merely a dense infiltration of lymphocytes and polymorphonuclear leukocytes with total disappearance of the normal muscular tissue; in others the normal tissue is moderately well-preserved, but thoroughly infiltrated with lymphocytes, young connective-tissue elements, and newly formed capillaries. The endocardium of the portions of the heart involved in this process is greatly thickened, both the endothelium and the subendothelial layer participating in the thickness, which is due to a proliferation of endothelial and connective-tissue elements without the signs of an acute inflammatory process. The pericardium presents no pathological features. A few of the smaller bloodvessels show slight endothelial proliferation. Aside from this, their study reveals no abnormalities.

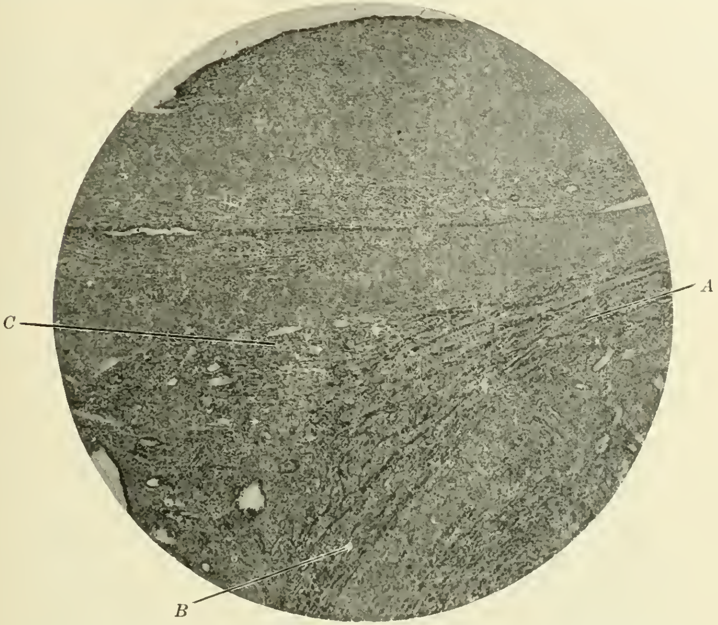

Fig. 7.—Photomicrograph of the bifurcation of the bundle of His. *A*, undivided portion; *B*, the branch to the right ventricle; *C*, the branch to the left ventricle.

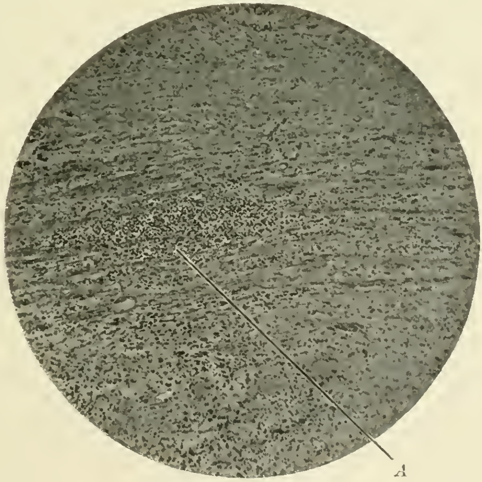

Fig. 8.—Photomicrograph, showing an area of round-cell infiltration in the bundle of His.

Diagnosis. Gumma of the heart; acute vegetative endocarditis; chronic passive congestion of the spleen, liver, kidney, intestines, stomach, and lungs; œdema of the lungs; angioma of the liver; bilateral pleural transudate.

Histological Examination of the Auriculoventricular Bundle of His. For histological examination of the auriculoventricular bundle of His, a section of the heart corresponding to the following boundaries was excised: Considering the heart to be placed vertically, with the ventricular septum anteroposteriorly, the anterior boundary corresponded to the middle of the right aortic leaflet; the upper boundary, to a line drawn parallel with the free margin of the aortic leaflets 8 mm. below the uppermost portion of their attachment; the lower boundary, to a line drawn parallel to the upper boundary 1.4 cm. below the lowest point of insertion of the posterior aortic leaflet; the posterior boundary, to the posterior margin of the interauricular septum. The anterior leaflet of the mitral valve and a portion of the median tricuspid leaflet were then cut away from this block. Serial sections in a plane at right angles to the long axis of the heart were cut from this block, commencing at its inferior surface. The sections were 10 microns in thickness. Each sixth section was stained with hematoxylin-eosin; each seventh section with hematoxylin-van Gieson. Subsequently the intervening sections of the first one hundred and fifty were also stained with hematoxylin-eosin.

As the histological findings involving the bundle of His are limited to a series of thirty-nine consecutive sections, we will confine our attention to them. Numbering the uppermost one of these, that is, the one nearest the auricles, 1, the numbers will increase as we approach the apex of the heart. Though in some of the sections above No. 1, portions of the bundle are visible, it is here that it becomes evident throughout its course from the auricular septum to its bifurcation into a right and a left branch. The macroscopic appearance of this section is shown in Fig. 6, in which the course of the bundle, just visible macroscopically in the section, is shown running in a curved direction from slightly above the auriculoventricular septum toward the left ventricular surface of the interventricular septum, then just beneath the endocardium to the point of bifurcation A. Fig. 7 shows the bifurcation into the branch for the right ventricle and the branch for the left ventricle. Though the course of the bundle throughout the first seventeen sections is easily traceable, it is microscopically by no means intact. Especially throughout the undivided portion there is an extensive round-cell and young connective-tissue infiltration. Some of these areas are shown in Figs. 8 and 9. As Section 18 is reached, a much more extensive involvement of the bundle is observed, resulting in a complete solution of its continuity. Here the gumma shown at G in Fig. 10, which is a reproduction of Section 18, has extended sufficiently to involve the bundle at the point H, Fig. 10, resulting in a complete disappearance of the muscle fibers and their substitution by the infiltrating tissue of the gumma. This interruption in the continuity of the auriculoventricular bundle is shown in Fig. 11. Photomicrographs of the terminations of the two fragments are shown in Figs. 12 and 13. Throughout the remaining sections in which the bundle is visible this interruption persists. Finally, arriving at a level below that of the undivided portion of the bundle, only the right and left branches can be seen, the left lying just beneath and parallel to the endocardium of the left ventricle, the right showing as a small bundle cut in cross-section lying nearer and nearer to the right ventricular surface of the septum, as we descend toward the apex of the heart.

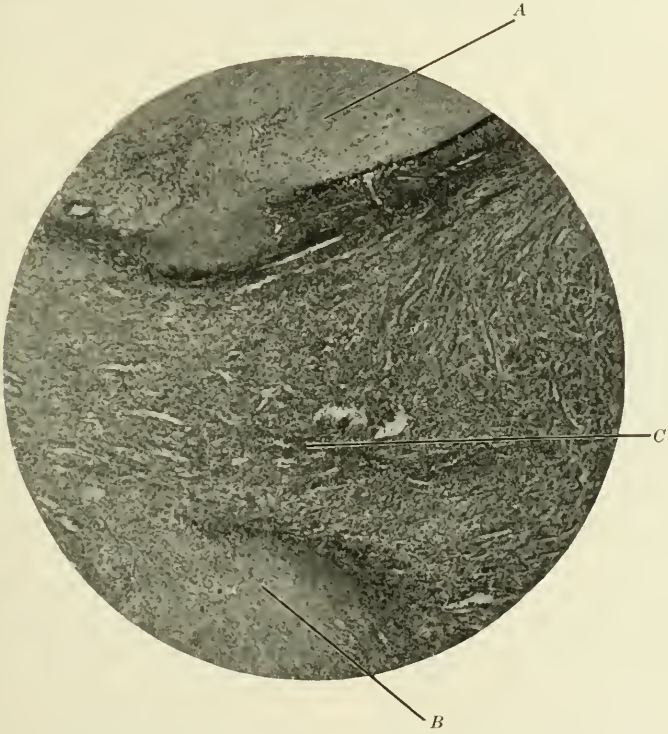

Fig. 9.—Photomicrograph showing an area of round-cell and spindle-cell infiltration in the bundle of His, just as it emerges from the interauricular septum. *A*, auriculoventricular septum; *B*, base of middle tricuspid leaflet; *C*, round-cell and spindle-cell infiltration in bundle of His.

Though numerous theories have been proposed for the cause of heart-block in the human being, almost all have been without pathological basis. From the recent work in the physiology and anatomy of the heart, we now have every reason to believe that a definite anatomical lesion, involving the bundle of His, is responsible for this condition in man. On account of the absence of examinations for lesions in the bundle of His in the early pathological reports of cases of Adams-Stokes disease, their consideration in the light of our present knowledge can be practically ignored. The findings looked upon as causal in these cases were probably purely coincidental.

A number of cases have been recently reported with more or less definite pathological lesions involving the auriculoventricular bundle. Stengel has reported a typical case in which there were macroscopically fibrous and calcareous processes at the base of the anterior mitral leaflet involving the area traversed by the bundle of His. Schmoll's[39] case showed scar formation in and around the bundle of His leading to atrophy of the muscular elements of the bundle and separation of its fibers from the ventricular muscle. Handford[40] reports a case in which the autopsy revealed gummas, involving the auriculoventricular septum. He does not report the histological examination of the bundle of His itself. The case of Jellick, Cooper, and Ophüls[41] showed anemic necrosis of the muscular septum in the region of the bundle of His consequent upon recent thrombosis of its nutrient arteries. The autopsy of Grünbaum's[42] case dis-

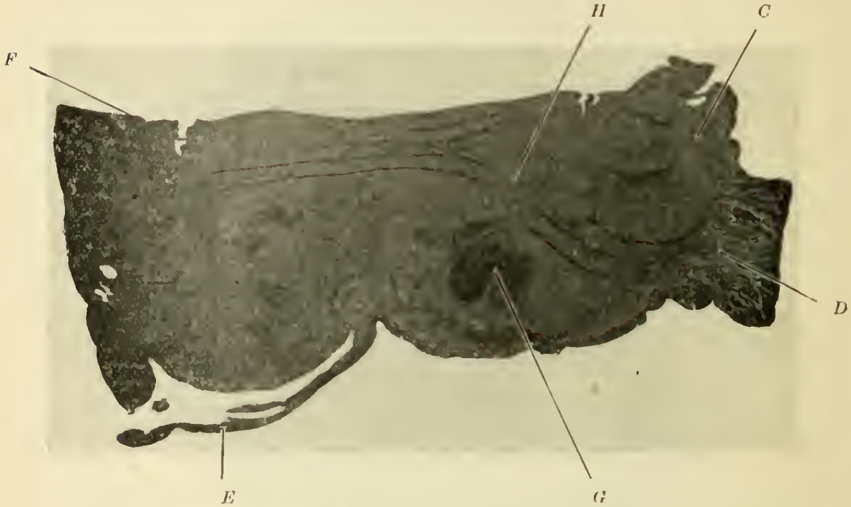

Fig. 10.—Photograph (slightly enlarged) of Section No. 18, with a tracing, showing the location of the interruption in the bundle of His; *H*, location of the interruption in the bundle of His; *G*, the necrotic center of the gumma destroying the bundle at the point *H*; *C*, *D*, *E*, and *F*, as in Fig. 3.

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[39] Dent. Arch. f. klin. Med., 1906, lxxxvii, p. 554.

[40] Brit. Med. Jour., December 31, 1904.

[41] Jour. Amer. Med. Assoc., 1906, xlvi, v. 361.

[42] Quoted by Ewart, Progressive Medicine, September, 1906, p. 74.

closed a gumma of the interventricular septum. Keith and Miller[43] report a case which was apparently one of Adams-Stokes disease in which there was found cicatrization of a gummatous mass involving all of the upper portion of the bundle of His and in addition gumma of the coronary sinus, obliteration of the superior vena cava and partial occlusion of the coronary arteries. Sendler[44] saw a case in which a cartilaginous tumor of the interventricular septum was found. The reports of most of these cases have not included the histological examination essential to the determination of the lesion in so delicate a structure as the bundle of His.

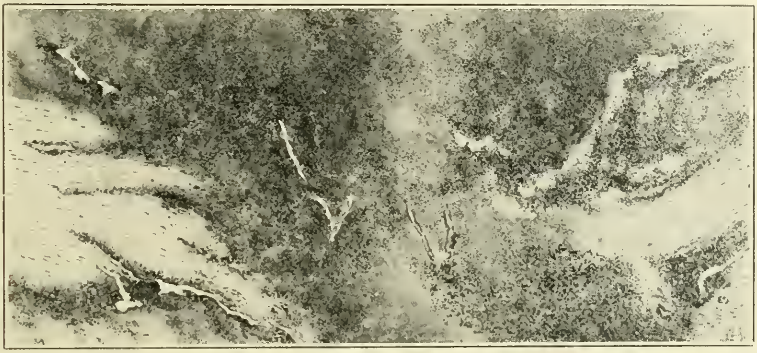
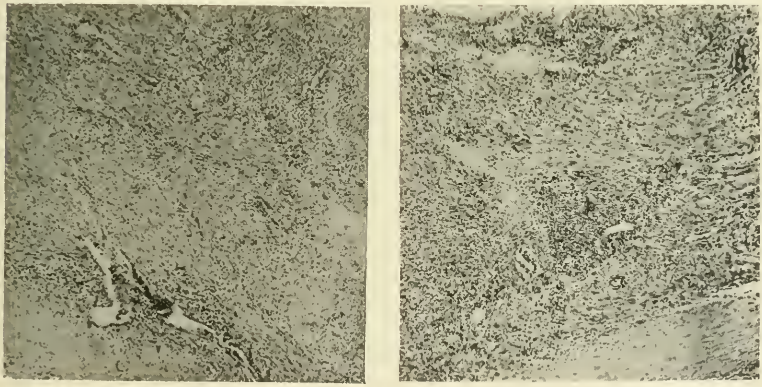


Fig. 11.—The interruption in the continuity of the bundle of His. The two fragments of the bundle are seen entering at the right and left sides of the illustration. Between their two terminations is tne infiltrating tissue of the gumma shown at *G*, in Fig. 4. Photomicrographs of the terminations of the two fragments are shown in Figs. 12 and 13.



Figs. 12 and 13.—Photomicrographs of the terminations of the two fragments of the bundle of His, as shown in Fig. 11.

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[43] Lancet, November 24, 1906.

[44] Cntrbl. f. klin. Med., 1892, p. 642.

The association of syphilis and Adams-Stokes disease appears to have been a peculiarly frequent one. Aside from the cases of Handford, Keith and Miller, and Grünbaum, quoted above, Jacquet, Osler, and Erlanger report cases in which there was clinical evidence in support of the view that syphilis was responsible for the condition. Erlanger and Jacquet mention this frequency of association, but the latter apparently assumes some obscure relationship between the two rather than the actual gummatous involvement of the bundle, as occurred in our case and apparently also in some of the earlier cases reported.

As in all other pathological conditions, the prognosis in Adams-Stokes disease depends upon its cause. First of all, we must clearly bear in mind that all cases of marked bradycardia are not necessarily instances of Adams-Stokes disease. Bradycardia may result from a great number of causes. Thus, it may occur physiologically from hunger, fatigue, pregnancy, and from individual or family idiosyncrasy. Clinically it is met with during the convalescence from infectious diseases, such as pneumonia, typhoid fever, influenza, diphtheria, erysipelas, etc. Riegel has noted the symptom in many cases of gastrointestinal disturbance, gastric ulcer, chronic gastritis, carcinoma, and jaundice. A slow pulse may also be encountered in diseases of the respiratory tract, notably emphysema, and in disease of the heart or bloodvessels—coronary sclerosis, myocarditis, atheroma of the aorta, and embolism of the coronary artery. It occurs also in renal disease—uremia, hematuria. Occasionally it has been noted in diabetes, chlorosis, or other forms of anemia. Bradycardia also occurs in diseases of the nervous system—epilepsy, apoplexy, meningitis, tumors of the brain, diseases of the medulla, and cervical spinal cord, mania, paresis, and melancholia. It has been reported in diseases of the genital system, and of the skin. Finally comes the important toxic group—tobacco, coffee, alcohol, lead poisoning, digitalis, etc.[45] [46]

So much for bradycardia simplex. In order to make a diagnosis of Adams-Stokes disease we must have not only the slow pulse, but also the increased pulsation of the veins, and the nervous symptoms. It seems quite reasonable to suppose from what we now know of the pathology of the bundle of His that minor grades of auriculoventricular dissociation may exist for years without giving rise to marked symptoms. In other words, the degenerative changes in the bundle are so slight in extent, and so slow in progression, that the patient dies from some other cause before the typical symptoms of Adams-Stokes disease develop. Doubtless the combined studies of the arterial and venous pulses which are now being made by different observers with the Jacquet cardiosphygmograph, the

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[45] Grob, Deut. Arch. f. klin. Med., xlii.

[46] Babcock, The Heart and the Arterial System, 1903, p. 624.

Mackenzie polygraph, or other instruments, will in the near future throw much light on this and many other hitherto obscure cardiovascular diseases.

Since it has already been shown that several cases of Adams-Stokes disease have been due to gummas involving the bundle of His, heroic doses of the iodides, if resorted to at a sufficiently early stage, may bring about a cure. This has apparently been accomplished in one case reported by Jacquet and Erlanger. In cases in which the lesions are sclerotic or necrotic in character no expectation of recovery can be entertained and they offer practically no hope of other than a fatal issue.

From the accumulated evidence of those cases of Adams-Stokes disease in which satisfactory tracings have been made it seems that the cases were due to heart-block. Now the question arises, May heart-block exist without producing the symptoms of Adams-Stokes disease? Belski and Mackenzie have reported cases of auriculoventricular dissociation without the symptoms of Adams-Stokes disease other than bradycardia. It follows, therefore, that there must be another factor concerned in the production of the symptom-complex. In all probability the ventricular asystole, which is the cause of the syncopal attacks, is brought about by the sudden occurrence of complete heart-block, for as Erlanger has shown, when the bundle of His is suddenly compressed, ventricular activity is in abeyance for a considerable time, that is, for the time required by this structure to inaugurate its own rhythm. This explanation, of course, is applicable only to cases of partial heart-block. Cases such as ours, in which the block is complete, cannot be accounted for in this way. Schmoll has suggested that in the latter event a condition of ventricular depression of conductivity exists, analogous to that which is present in the bundle of His, while the stimulus production goes on normally. If such a state of affairs existed we should expect to find a definite relationship between the rate of the ventricular contractions before and after the block took place. This has actually been noted in a few cases. Thus in His' case the pulse-rate fell from 36 to 18, and later returned to 36. Once more it fell to 12, and subsequently rose to 24, indicating that the original rhythm was undisturbed, but that it required more than the normal number of stimuli to provoke a response. Similar observations have been made by Erlanger, Cornil, and Schmoll, and are, of course, the exact reverse of those noted by Hoffman[47] in cases of paroxysmal tachycardia.

In conclusion, we desire to express our indebtedness to Mr. E. B. Krumbhaar, a student in the University of Pennsylvania, for assistance in the histological examination of the heart.

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[47] Ztsch. f. klin. Med., vol. xliii, p. 206.

THE PATHOLOGICAL PHYSIOLOGY OF CHRONIC ARTERIAL HYPERTENSION AND ITS TREATMENT.

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The Pathological Associations of Hypertension. Persistent high blood-pressure is a striking departure from the physiological norm of the circulation, and, though only a symptom, and in nowise a clinical entity, yet is itself a cause of such important secondary lesions in the heart and vessels, that it deserves independent consideration.

It is common in nephritis, especially of the chronic interstitial type. It occurs occasionally in arteriosclerosis. In addition it constitutes the chief feature, during life, of a clinical group of cases, which present the same cardiovascular lesions as chronic Bright's disease, but no evidence of renal involvement. In a recent analysis[1] made by me of one hundred and thirty cases showing a systolic pressure over 200 mm. seen in Dr. E. G. Janeway's office practice, 13 per cent. belonged in this category.[1] Autopsy experience bears this out, both my own and that of observers whose competence is evident.[2]

This group, formerly described by v. Basch as latent angiosclerosis, is now named by the French "hypertension arterielle," to which certain German writers object strenuously. It is perhaps better classed as "hypertension of unknown origin," or "hypertension without nephritis." The idiopathic hypertrophy of the heart of the older pathologists, undoubtedly expressed the same observation, from a standpoint one remove further from the primary lesion.

Its Physiological Mechanism. (*a*) *In Arteriosclerosis.* The hypertension of arteriosclerosis finds the readiest analogy in experiment on the living animal. When the cervical cord has been cut, and reflex vasoconstriction excluded, ligature of the abdominal aorta will raise blood-pressure only when above the origin of the cœliac axis.[3] In accord with this are Hasenfeld's[4] and Hirsch's[5] observations on cardiac hypertrophy. In arteriosclerosis they found this only when there was involvement of the splanchnic vessels, or the thoracic aorta. That the conditions are far from simple is evident, since Marchand[6] has adduced many similar cases in which hypertrophy was lacking. His observations were not by Müller's method, but I think few pathologists will dispute them. My belief is, that the nutritive factor in the production of hypertrophy should receive more consideration. In arteriosclerosis especially, changes in the coronary arteries and the degenerative tendencies of age make a poor basis for the development of new cardiac muscle. One must also bear in mind the demands made on the circulation by the individual, and the possibility of atrophy in a previously hypertrophied heart, after prolonged physical and mental rest in a hospital ward. Autopsies show terminal pictures, which are not a secure basis for too wide inference as to conditions which may have obtained years before.

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[1] Amer. Jour. Med. Sci., 1906, cxxxi, 772.

[2] Krehl, L., Deut. med. Woch., 1905, xxxi, p. 1872.

[3] v. Bezold, Untersuchungen über die Innervation des Herzens. Leipsig, 1863, pp. 223–229, and Ludwig und Thiry, Sitszungsber. d. Kais. Akad. d. Wiss., math. naturw. Cl., 1864, vol. xlix, 2, p. 442.

[4] Deut. Arch. f. klin. Med., 1897, vol. lix, p. 193.

[5] Ibid., 1900, vol. lxviii, p. 55.

Certainly in the small number of patients in whom arteriosclerosis leads to persistent high blood-pressure, the latter must be a physiological necessity for the maintenance of an adequate abdominal circulation.

(*b*) *In Nephritis.* The problem of nephritic hypertension is much more complicated. Physiological experiment has sufficed to put out of court the simpler mechanical theories, such as Traube's, since ligation of the abdominal aorta just above the renal arteries does not raise blood-pressure. The simpler chemical theories of stimulation of the heart and vasomotor mechanisms by retained metabolic waste products (Johnson) also must be abandoned, since hypertension is most frequent in that type of nephritis in which the least retention occurs.

The anatomical lesions found in the smaller arteries seem adequate to produce the hypertension in many of the chronic cases which come to autopsy. I believe that thickening of the intima and muscularis, throughout the splanchnic arterioles, plays a large part in the perpetuation of the hypertension, just as cardiac hypertrophy does; but it may be, and probably is, equally its result. Hypertension has been observed within twenty-four hours of the onset of acute scarlatinal nephritis,[7] when anatomical changes in heart and vessels are out of the question.

A purely anatomical increase in peripheral resistance is extremely improbable, in the light of the prompt response to vasodilators in many of these cases. For instance, a woman in my wards at the City Hospital, in whom neither nitroglycerin nor anything else had the slightest effect on the average level of blood-pressure, responded to three doses of nitroglycerin of $\frac{1}{50}$ grain each, five minutes apart, given for the relief of nasal hemorrhages, by a drop from 195 to 95 mm. Any explanation, consonant with such facts, must recognize increased vasomotor tone as a predominating factor.

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[6] Verhandl. d. 21ster Kongr. f. in. Med., 1904, pp. 54-58.

[7] Butterman, Deut. Arch. f. klin. Med., 1902, lxxiv, 1.

Increased viscosity of the blood has been suggested as an explanation, but has failed of proof in cases examined.[8] The return to one of Bright's original suggestions, that primary stimulation of the heart may be the cause of its hypertrophy, as Senator[9] advocates, need not detain us in discussing hypertension, for a hypertrophied heart alone cannot produce highblood-pressure, in the presence of a normal splanchnic circulation. This is evident from our knowledge of the regulatory powers of the splanchnic vasomotor system, and comes out clearly in such experiments as Israel's.[10]

Take it all together, a theory recently advanced by Loeb,[11] from Krehl's clinic, seems to accord with more ascertained facts than any other. Observations on the relation of glomerular changes to hypertension, both in acute and in chronic cases, convinced him of the striking parellelism of the two. Most observers would, I think, agree with him, that hypertension, or cardiac hypertrophy, is seen with those types of nephritis in which the glomeruli are extensively diseased (we must except amyloid disease, of course, in which the circulation within the glomeruli may not perhaps be so much interfered with, and in which the nutritive conditions are insufficient for the development of added heart force). The functional activity of the kidney is dependent on the quantity of blood flowing through the glomeruli in a given time. Normally, increase in the rate of flow can be produced by local vasodilatation, as in all glands. With marked increase of capillary resistance within the glomeruli, however, it is unlikely that this should suffice; therefore, Loeb postulates a stronger demand from the glomeruli, which goes beyond the local vasomotor system of the kidney, and, reaching the cerebrospinal centres, calls forth a reflex splanchnic vasoconstriction which increases the general blood-pressure sufficiently to maintain a normal rate of flow through the diseased glomeruli. Such a reflex splanchnic vasoconstriction evoked by the glomeruli, when coupled with a more gradual development of permanently increased peripheral resistance and heart force, through secondary hypertrophy of the arterial wall and the heart muscle, and subject to augmentation, as metabolic end products accumulate in the blood and call for still greater functional activity from the remaining kidney tissues, seems to me very plausible.

HYPERTENSION OF UNKNOWN ORIGIN. Clearly little can be said in these cases. Krehl[12] has observed extensive lesions in the brain arteries, and Loeb suggests the possibility of a vasoconstrictor reflex from this and other organs similar to that hypothecated to explain nephritic hypertension. For the brain, our knowledge of the enormous rise in pressure produced by anemia of the medulla (Kocher, Cushing) and of the dependence of the brain circulation upon the general blood-pressure make this far from improbable.

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[8] Hirsch u. Beck, Deut. Arch, f. klin. Med., 1902, lxii, p. 560.

[9] Die Erkrankungen der Nieren, Wien, 1902, pp. 110–129.

[10] Virchow's Arch., 1881, lxxxvi, 299.

[11] Deut. Arch. f. klin. Med., 1905, lxxxv, p. 348.

[12] Deut. med. Woch., 1905, xxxi, p. 1872.

Hypertension a Compensatory Manifestation. Whatever be the exact mechanism of its production, we cannot avoid the conclusion that hypertension expresses an attempt of the organism to maintain an adequate speed of capillary flow through the kidney or other important organ, which would be impossible without it. True, it is a symptom of disease, but still more truly is it a proof of the body's defensive reaction against the disease. This conception is the foundation of any rational treatment.

Effects and Dangers of Hypertension. Nevertheless, there is quite a different aspect to the matter. Persistent high blood-pressure, however necessary, must be followed by important changes, both in structure and function, of the circulatory organs. Hypertrophy of the left ventricle is the most obvious consequence, but many so-called arteriosclerotic changes of Bright's disease are probably equally secondary. Functionally, the circulation at high pressures shows several marked deviations from the normal. The diminishing distensibility of the arteries, as the pressure within them rises, causes an increasing systolic elevation of pressure and suddenness of the pulse wave, which greatly favor arterial rupture at any weakened point. Then, too, the vasomotor regulation is impaired, and ordinary physiological rises in pressure tend to become exaggerated, often bringing attacks of anginal pain in their train. In addition crises of hypertension may appear from causes little understood, as in acute uremic seizures.

The danger most to be anticipated is the gradual development of insufficiency of the left ventricle. Then inadequate capillary circulation, on the one hand, intensifies the primary trouble; and pulmonary venous stasis, on the other, brings dyspnœa on exertion, cardiac asthma, or œdema of the lungs in its train. Later, if the right heart also fail, general venous stasis dominates the picture, and the arterial pressure falls, sometimes to a striking degree.

The Therapeutic Indications. From the consideration of these two aspects of hypertension, as a compensatory effort, and at the same time a disturbing factor in the circulation, three lines of treatment suggest themselves: Preventive, adjuvant, and emergent.

(*a*) *Preventive Treatment.* Preventive measures must be directed mainly toward limiting or ameliorating the primary disease, and hence, its effects on the circulation. They are comprised in the general hygienic and dietetic treatment of chronic nephritis. The effect on the blood-pressure is often striking, especially that of rest and a milk diet in the exacerbations of nephritis, so common in our hospitals. Equally good results often follow the more difficult readjustment of diet and mode of life in middle-aged high livers, who are in the early stage of the disease, a return of the blood-pressure to nearly normal limits being possible. Allbutt has preached this insistently. Massage belongs among the preventive measures and often seems of value. The iodides, I think, must also come in this category. More definite measures, from the standpoint of the blood-pressure, are the avoidance of all markedly hypertensive influences, such as excessive or unusual physical or mental work, straining at stool, and the excessive use of tobacco.

(*b*) *Adjuvant Treatment.* The adjuvant treatment of hypertension is beginning to receive the recognition it deserves. Formerly the discovery of high blood-pressure was too frequently the signal for the attempt to reduce it at any price. Fortunately, as Krehl has lately said, such efforts were usually unavailing. This failure of vasodilators to reduce the general level of pressure in such cases has usually led to loss of faith in the drugs, rather than to an appreciation of the struggle of the organism to maintain its life in spite of the physician. We begin to know the physiological necessity better, and to view with alarm any fall in pressure which is not the result of preventive treatment and the accompaniment of other evidence of improvement. As a rule, such a fall marks the onset of cardiac insufficiency and calls for digitalis, our mainstay in combatting asystole. Not even in mitral insufficiency do more brilliant results follow digitalis therapy than in the failing heart secondary to hypertension; and an increasing blood-pressure, though not invariable, for the conditions are complicated, usually presages a winning fight. The indications for digitalis, and its congeners, are the same with hypertension as without, and no blood-pressure reading should make us hesitate to administer it.

(*c*) *Emergent Treatment.* Emergent treatment includes all the means used to forestall or cut short those dramatic moments which may come without warning to any victim of hypertension when life suddenly hangs in the balance. Here the vasodilators find their proper place and rational use, and are as indispensable as is digitalis, when the indications are for adjuvant treatment. Nitroglycerin and the other nitrites are the most efficient for the relief of the anginal seizures which often follow exertion when the arterial pressure is high, and their action sometimes seems magical. In acute uremia, venesection fulfils the double purpose of relief of hypertension, through reduction of blood-volume, and removal of circulating toxic substances. Light chloroform narcosis, so commonly relied on to stop convulsions, produces a fall in blood-pressure.

Dangerous nasal hemorrhages, an occasional accident, may often be checked by nitroglycerin in full doses. With cerebral hemorrhage the conditions are much more complicated, since the hypertension, when intracranial tension is much increased, may be essential to the preservation of life, while at the same time favoring continuance of the bleeding. In many cases the hypotensive measures, bleeding and vasodilators, seem to do good. Probably in these the hypertension was pre-existent, and the intracranial pressure not much increased in the posterior fossa. That a differentiation is possible clinically between the hypertension indicating beginning anemia of the medulla oblongata and the hypertension which helped produce the arterial rupture, I do not believe, unless previous records of the case are at hand. The former must, under no circumstances, be interfered with.

Attacks of œdema of the lungs, always of great danger and most agonizing to the patient, do not admit of simple explanation or routine treatment. Unquestionably sudden weakness of the left ventricle[13] is a large factor in their production in the patients we are considering. Such an occurrence calls at least for the simultaneous use of rapid heart-muscle stimulants, and of vasodilators to reduce to a minimum the demands on the left ventricle.

In a certain number of patients in whom severe abdominal pains or intense headache was a prominent symptom, I have been struck by the beneficial effect of morphine, both on the general condition and on the blood-pressure. The removal of the reflex vasoconstriction, which pain produces, may explain it; but this factor alone has not always seemed to me adequate to account for the remarkable and sustained improvement in a few cases of this class.

In closing I would emphasize one point strongly. The height of the blood pressure gives no indications whatever for treatment, except along preventive lines. Hypertension is no more to be attacked therapeutically than is a heart-murmur. Only those symptoms which point unmistakably to inadequate maintenance of the circulation, or to sudden threatening of danger, give us the signal to interfere.

INORGANIC LATE-SYSTOLIC PULMONARY MURMURS IN INFANCY AND CHILDHOOD.[1]

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Two years ago at the meeting of the American Medical Association we presented to the Section on Pediatrics a paper on inorganic late-systolic pulmonary murmurs in children. We were stimulated to do this because our observations were not in accord with the generally expressed views in the literature relating to this subject, and especially because of the almost universal statement in the text-books on pediatrics that functional cardiac murmurs are extremely rare in children under four years of age, some authors even denying their existence.

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[13] Cohnheim and Welch, Cohnheim's Gesam. Abhandl., Berlin, 1885, p. 593.

[1] Read at the meeting of the American Pediatric Society, 1906.

In so far as we have observed, the views expressed in the text-books have not changed, and, therefore, we have decided to offer a paper on the same subject to this Society in order to elicit the views of its members as to the frequency of functional cardiac murmurs, the nature of their origin, and their significance. It seems to us that the question is one of great importance, on account of its relationship to the subject of organic disease of the heart, and one concerning which this Society might do well to express an official opinion.

We do not presume to offer anything new, but wish merely to reiterate our impressions as to the frequency of this murmur and our interpretation of its meaning. We have little doubt that it is commonly recognized by pediatrists, but we feel that the text-books, which serve as a medium for educating the profession at large, should be correct in their statements regarding it. Speaking from experience, we are inclined to believe that the average practitioner not only considers this murmur very infrequent, but constantly mistakes it for an indication of organic disease.

From the descriptions in the literature we feel justified in assuming that all murmurs described as functional, inorganic, or anemic, being systolic in time and having their point of maximum intensity in the pulmonary area, are identical with the murmur which we are discussing. The cardiopulmonary and the diastolic functional murmurs and the functional regurgitations, especially the two former, we have found extremely uncommon in early life, and we do not believe that they should ever be confounded with the murmur to which we refer.

It seems a somewhat curious fact that a number of the French and German writers consider the cardiopulmonary murmurs quite common in infancy and early childhood, and that some of them evidently confound them with the type of murmur under discussion. It is especially interesting to note that Hochsinger, who makes such positive statements regarding the so-called "anemic murmurs," is at the same time of the opinion that the cardiopulmonary murmurs, whilst very frequent after the second year, are nevertheless observed during the first year and that this murmur "is a frequent phenomenon in infancy."

There is evidently a diversity of opinion as to what constitutes a cardiopulmonary murmur. The impression we have gained of this murmur is that it is distinctly systolic in time; that it is heard with maximum intensity at the apex and along the left border of the heart; that it has a tendency to greater transmission to the left;

that it is short and whiff-like in character and lacks the soft, prolonged, blowing sound of the late systolic murmur heard in the pulmonary area. The murmur further differs in that it is heard most distinctly toward the end of inspiration, and in that it often disappears entirely at the end of the forced expiration, especially when the patient is recumbent.

Characteristics of the Late-systolic Pulmonary Murmur. The murmur we wish to describe does not occur with the first sound; the first sound is always distinctly audible and the murmur is superadded, as it were, sometimes with a definite interval of silence, and is not uncommonly, especially when it is not loud, mesosystolic in time. This is one of the principal characteristics of the murmur, and unless one studies the systolic interval carefully the soft indistinct forms will be readily overlooked.

This murmur is ordinarily soft-blowing in character; moderately high-pitched; occasionally almost musical, and more rarely low-pitched and rather rough. It varies in duration, sometimes occupying the greater part of the systolic interval, but more frequently its early portion.

Its point of maximum intensity is most often the second left interspace close to the sternal border. It is sometimes best heard a little more to the left and quite frequently over the third costal cartilage; it sometimes has a second point of increased intensity, especially when the patient is examined in the sitting or standing posture, within and above the apex, about midway between the midclavicular and parasternal lines in the third or fourth spaces, depending upon the position of the apex.

The *position of the patient*, the *respiratory act*, and *exercise* have a striking influence upon this murmur. It is always greatly increased in the recumbent posture and frequently heard only when the patient is recumbent; it should never be considered absent unless the patient has been examined in the recumbent, as well as the upright, position. It is best heard at the end of the expiration and sometimes only heard when the breath is held at the end of a forced expiration. It will often disappear when the lungs are filled with air. *Exercise* exaggerates it. It is not uncommon to have the milder murmurs entirely disappear after the patient has remained recumbent for a time, to return again after the patient engages in some mild exercise.

There is great diversity of opinion as to the character of the second pulmonic sound associated with this murmur. In our experience, it has been invariably accentuated, and we do not, therefore, place any dependence upon the character of the second sound as an aid in the differentiation between this and organic murmurs of the heart.

Ordinarily, the murmur is not transmitted over a very wide area. It is occasionally audible only over the second interspace, but more commonly also over the third and fourth spaces, in or within the parasternal line, just above and within the apex of the heart, and, when it is very loud, it may be heard over the body of the sternum, at the apex-beat, and occasionally even beyond it. Sometimes a murmur similar in time and quality may be heard in the aortic area, but whether this murmur is the same murmur transmitted, or whether it is a different murmur produced by the existence of similar conditions in the aorta, we have not been able definitely to determine. When the murmur is audible over this wide area, it always continues to have its point of maximum intensity in the second left interspace.

The *position of the apex-beat of the heart* is not altered and the *area of heart dulness* is not increased.

There is almost invariably associated with this murmur a loud venous hum over both sides of the neck, commonly audible also below the sternal ends of the clavicles, sometimes over the manubrium of the sternum, and less frequently in the aortic area. In addition to the venous hum, one can quite frequently hear systolic blowing murmurs over the carotid and subclavian arteries.

The *relationship of anemia to this murmur* has been freely discussed in the literature and there exists a great diversity of opinion regarding it. We do not believe that anemia *per se* is responsible for its production, but, unquestionably, the conditions giving rise to the murmur are frequently associated with anemia.

In the series of eighty cases herein reported, we have determined the hemoglobin content by means of the Dare apparatus in all but eight. In three instances, the hemoglobin content was below 60 per cent. There were twenty-three which showed no functional murmurs. The hemoglobin content in these cases was respectively: 65, 70 twice; 71, 75, 78, 79, 80 five times; 81, 83 twice; 88 three times; 89, 90 twice; 92 and 94; whilst in those showing the murmur, there were found such percentages as 80 twice; 83, 85 five times, 87, 88, 90 twice; 92 and 100. There were, however, among these latter, a number of cases showing lower hemoglobin content, as many as 11 having less than 70 per cent. It will be seen from these figures that in very few instances was there a marked degree of anemia, and in one case the murmur was present when the hemoglobin content was 100. Quite often we have failed to find these murmurs present in infants suffering from severe forms of anemia.

We are unable to offer a new theory for the cause of these murmurs. The number appearing in the literature would seem pretty effectually to cover the possible modes of production. There is practically no abnormal condition concerning which more speculation has been indulged in. We have been impressed by the apt description which Balfour gives of the pulmonary area when he refers to it as the "region of romance." We will not discuss all of the theories which have been advanced, but limit ourselves to the consideration of those which seem more or less plausible.

We wish, first of all, to cite the view expressed by Leube. He believes the point of production of these murmurs to be at the base of the heart, in the beginning of the great vessels, especially the pulmonary artery, the point at which they are most distinctly heard. He says: "The wall of the pulmonary artery, being in an atonic condition, dilates more markedly in the systolic pressing in of the blood than under normal circumstances; by this dilatation of the beginning of the pulmonary artery, isochronous with the systole, there are created all of the conditions necessary for the production of the murmur." According to this conception, the time of the murmur would coincide with the time of expulsion of the systole. This represents the second part of the systole and corresponds to the first part of the descending line of the cardiogram, and, therefore, the slight sound which is formed during the systolic closure may be heard for a brief period besides the murmur; that is, it introduces the latter.

R. Geigel's explanation of this murmur is that it is produced by a relatively high ventricular pressure and an abnormally low pressure in the aortic and pulmonary arteries, which prevents the closure of the semilunar valves during the so-called closure period, giving rise to the production of a murmur at the base of the heart, the area of physiological stenosis. Leube is inclined to believe that murmurs sometimes occur in the aortic and pulmonary areas which occupy the entire period of the systole and which can be explained on the basis of Geigel's theory, but that this is not satisfactory for the explanation of the systolic murmur in which the sound of the systolic closure is audible.

Cabot accepts as the most plausible explanation that, as regards most functional murmurs, they are due to a temporary or permanent dilatation of the conus arteriosus or to pressure or suction exerted upon the overlapping lung margins by the cardiac contractions.

Fagge explains them thus: The basis of the two main arteries are supposed to be unable to retreat in correspondence with the diminished volume of the blood in anemia to the same extent as the orifices through which the blood enters them, in consquence of which vibrations occur in the current.

Drummond believes the murmur to be "the result of vibrations generated in the conus arteriosus of the right ventricle, at least in the cases in which its maximum occupies the second and third interspaces close to the sternum on the left side."

Clifford Allbutt "leans to the belief that the solution of the causation of this murmur will be found in some altered relation between the blood and the walls of the vessels, especially the pulmonary artery and conus arteriosus, so that an excessive vibration of the walls takes place."

Rudolf thinks the murmur is caused by the pulmonary artery becoming dilated whilst the orifice remains normal, the blood then being split up into fluid veins as it passes from the ventricle into the vessel. He does not believe that the dilated conus arteriosus can of itself produce a murmur, but that the pulmonary artery must be dilated at the same time. Otherwise, one merely has a cavity opening into a cylinder which will not give rise to a murmur.

Nixon explains the mechanism of production of murmurs, only audible when the patient is recumbent in those who are not obviously anemic, as due to a diminution of tension in the artery, which allows a twist or contraction to be formed at its root during the ventricular contraction. In this event, the disappearance of the murmur when the erect position is assumed would be explained by the traction made upon the pulmonary artery by the weight of the heart tending to preserve the natural caliber of the vessel.

La Salle has noted that the administration of trinitrin and other vasodilators tends to augment functional murmurs, which corroborates the view that lowered arterial tension plays a large part in their production.

We quote Foxwell's explanation at some length because it seems to combine the views before mentioned and to interpret them in a very rational manner. He says: "We may consider the whole right ventricle as one-half of a cone, which has been described from apex to base. The stout septum ventriculorum forms the triangular surface of the section, and, as the left ventricle does not fail, no more does this septum; hence, we may look upon the ventricle as a triangular board to which is attached a semicircular convexity of rubber. It is evident that this yielding rubber will stretch most where it is least restrained by the rigid triangle, namely, along the semicircular border of its base. Also, that the portion of this border which will stretch most will be the conus, for the lowest part is held in check by the unyielding rigidly fixed inferior vena cava and the middle portion by the chordæ tendinæ of the tricuspid valve and their stout musculi. Further, the direction of the blood current is from the apex toward the pulmonary valves; the conus will tend to give, therefore, most in the direction where it is longest distended with blood.

"Given this dilatation of the conus, that of the pulmonary artery is easily explained. The dilating conus carries the valves upward and this shortens the length between the two extremities of the artery. The artery becomes lax and when distended with blood enlarges in its transverse diameter, so that its wall may occupy the same superficial area. Finally, we must remember the easy distensibility of the pulmonary artery, which is proportionately, after due allowance is made for the different tensions at which they work, some six times that of the aorta."

This explanation appeals to us as one of the most rational given and in a way accounts for the relatively greater frequency of these murmurs in early life. We know that the arterial tension is lower in children than in adults, and it seems reasonable to suppose, therefore, that conditions having a tendency to depress the arterial tension would make a more rapid and profound impression upon the circulation in early life and that there would, therefore, result a greater tendency to dilatability of the great vessels. Aside from this, it is not unlikely that the higher position of the diaphragm in early life, dependent upon the relatively large liver, the tendency to distention of the abdominal organs, and the undeveloped condition of the lungs, by maintaining the heart in a high position, relieve the tension on the pulmonary artery, to some extent "shorten the length between the two extremities of the aorta," thus rendering it more dilatable.

That the murmur is produced more frequently in the pulmonary artery than in the aorta is probably explained by the fact that the walls of the pulmonary artery are normally thinner and therefore more dilatable than those of the aorta, and by the fact that the resistance to the flow of the blood expressed from the heart is greater and more proximate in the pulmonary artery than in the aorta.

Frequency of Functional Cardiac Murmurs. Of the American text-books, Holt's is the only one which seems to appreciate the frequency of the existence of these murmurs, and we feel that the statements in his work do not do full justice to this phase of the subject. He says: "Accidental murmurs may be due to anemia and other blood conditions and, although not so common as in older patients, they are by no means rare even in infants." He further states that anemic heart murmurs are frequently heard in marasmus.

Koplik quotes Hochsinger as not having found a single anemic cardiac murmur in an examination of two hundred anemic children under four years of age. He states that he has, under exceptional anemic conditions, found a mild, blowing, basic murmur and that one such murmur occurred in a child under four years. He considers the "hemic murmurs" so common in the febrile conditions of adult life as "rarely heard, even in severe febrile affections with anemia in patients under three years." He has, however, heard them in children under three years of age with severe typhoid fever.

Rotch, in referring to this matter, says: "It is rare to meet functional cardiac disturbances before the middle period of childhood, but they increase in frequency as puberty approaches." He includes the inorganic murmurs in this statement. In the paragraph on the symptomatology of anemia infantum pseudoleukemica of von Jaksch, he says "there are often a venous bruit in the neck and functional cardiac murmurs."

Jacobi, in his text-book, says: "The functional murmurs are not so frequent in the child or adolescent as in the adult." In an article appearing in the *Transactions of the American Climatological Association* for 1900, he refers to a systolic murmur which he noted in an infant, aged thirteen months, suffering from an attack of pneumonia, which disappeared during convalescence. A fatal suppurative pleurisy following the pneumonia permitted a postmortem study of the heart and absolutely nothing abnormal was found in the organ. This he considered a functional mitral murmur.

Keating refers to functional murmurs in early life as "not uncommon."

Caille, in his text-book on *Differential Diagnosis and Treatment of Disease,* says: In children accidental murmurs are comparatively rare.

Cotton makes no reference to the frequency of inorganic murmurs. He says: "The soft, systolic bruit common to infectious fevers, the cardiorespiratory murmur of vesicular emphysema, and the before-mentioned sounds due to anemia and transient incompetency are all encountered in childhood."

Meigs and Pepper considered a certain percentage of the systolic murmurs heard over the body of the heart in chorea, functional in character and dependent upon the associated anemia.

Ruhräh makes the statement that hemic and functional murmurs are frequently heard in anemia and febrile diseases and are often mistaken for organic heart disease.

Churchill in discussing a paper by Butler read before one of the Chicago Medical Societies made the following statement: "At a recent Congress, Soltmann and Hochsinger said that 'hemic murmurs' in children under four years of age were extremely rare and that they were rare even up to eight years. If this statement be true, then there must be a deplorably large amount of organic heart trouble in children."

Morse in discussing the paper which we read before the American Medical Association made the statement that "functional murmurs are very common in infants."

The foregoing quotations represent the only printed statements made by past or present members of the American Pediatric Society, relative to this subject, which we have been able to find.

A few other American authors refer to the subject thus: Taylor and Wells say that the functional or accidental murmurs in childhood, particularly in older children, are of fairly common occurrence. Oppenheim believes that murmurs in children under two and one-half years of age are almost always congenital in origin, and that hemic murmurs are much more likely to occur in young children than those of organic disease.

The opinions expressed in the foreign text-books are very similar to those above quoted. The writers of German text-books seem to have been influenced by the statements of Hochsinger and Soltmann, both of whom, a few years ago, expressed the opinion that functional murmurs are extremely uncommon during the first three years of life and that they are not observed frequently until after the seventh or eighth year. Hochsinger even went so far as to state that all systolic murmurs heard over the precordium in children under four years of age should be considered organic murmurs.

Frühwald considers them rare in childhood, particularly during the first four years.

Graetzer considers them rare up to the seventh year of age and of very rare occurrence in children under four years of age, but admits their occasional existence even in nurslings. Baginsky makes no special reference to functional murmurs. In a section on anemia, he says: "Venous murmurs are audible in many cases, especially in later childhood."

Henoch believes the accidental cardiac murmurs very rare under four years of age.

R. Hecker and J. Trumpp embody the views of Hochsinger that "the so-called accidental inorganic heart murmurs in children under four years of age are of the greatest infrequency, and a systolic heart murmur with almost positive certainty proves the existence of an organic heart affection, even if it exists as the only provable heart symptom."

Filatou says that "anemic murmurs, although seldom, occur in children under three years of age."

Audeod, in his article in the *Traité de Maladies des Enfants,* says: "One does not find cardiac murmurs in anemic infants, but in the anemias of the second infancy cardiac bruits are common."

Among the English authors of text-books on Pediatrics, Dawson Williams and Ashby and Wright make no reference to the occurrence of these murmurs.

Goodheart and Still refer to them only as associated with chorea.

Hutchinson says that "a loud murmur heard over the precordium under two years is almost certainly congenital. Hemic murmurs may, however, occur in children under two years of age."

John Thomson says: "The functional murmurs are certainly much less common in children than in adults. They do occur, however, occasionally even in young children."

Donkin considers the occurrence of "anomalous and temporary murmurs over the precordia of healthy infants frequent.

In practically all other text-books on the diseases of children which we have consulted there is either no reference made to these murmurs, or if mentioned, the frequency of their occurrence, or the age at which they are most common, is not stated.

The opinions expressed in the journal articles and theses especially devoted to the discussion of this subject are quite similar to those found in the text-books, save that there are more authors who recognize their occurrence, the frequency of their occurrence, and the fact that they are common even in early infancy.

Garrod, for instance, states that murmurs identical in character with the hemic murmur in adults are met with even in very young children. He refers to an infant aged nineteen months in which such a murmur was present.

Looft reports a series of fifteen cases, ranging in age from three months to two and one-half years, only three being more than two years of age, in which such murmurs were present.

Rudolf found functional murmurs in 60 per cent. of the inmates of the Sick Children's Hospital in Toronto, while Butler found accidental systolic murmurs in 31.6 per cent. of a series of children between the ages of seven years and puberty. Sansom refers to the existence of these murmurs in early childhood. Weill holds the same views as Hochsinger as to their frequency.

Steffen considers accidental murmurs of more common occurrence under four years of age than is ordinarily believed.

Comby has found hemic murmurs at the base of the heart and in the vessels of the neck many times in anemic rachitic children.

Marfan thinks that anemia in young infants does not produce cardiac or vascular murmurs as frequently as in the adult, and he believes them very exceptional before the age of three years. He reports a case occurring in a boy, aged twenty-eight months, with symptoms of cardiac disease. The murmur was soft and systolic in time. The patient died and nothing abnormal was found in the heart.

Thiemisch reports a somewhat similar murmur in an infant aged six months. The autopsy revealed no abnormalities in the heart.

Drummond says that "cardiohemic" or "anemic" murmurs are quite common in children who are below par from any cause, especially when they are examined in the recumbent posture.

Practically all the authorities we have quoted admit the occurrence and comparative frequency in later childhood of functional murmurs of the type we describe, but we desire again to emphasize the fact that the vast majority, and especially the authors of text-books on pediatrics, consider accidental, anemic, or functional murmurs extremely uncommon prior to the fourth year.

In our former communication we had made no studies which gave us a very clear appreciation of the frequency of the inorganic late-systolic pulmonary murmur in infancy and childhood. We saw it often enough to make us realize that it is extremely common, and, in a study of a series of thirty-four consecutive cases in St. Vincent's Home, of infants ranging in age from two weeks to three and one-half years, we found it present in 21, or 61.7 per cent., distributed as follows: Eight times in nineteen children under one year of age; twelve times in fifteen children between one and three years of age; and in one child three and one-half years of age it was also present.

In order to obtain some additional evidence as to its actual frequency, we have studied forty-six children in the Southern Home for Destitute Children, ranging in age from three to ten years.

Of the five aged three years, four had the murmur; of the nine aged four years, seven; of the five aged five years, three; one of the five aged six years had an organic lesion and the four remaining had the functional murmur; five of the six aged seven years had it; three of the four aged nine years and only two of the five aged ten years.

It is seen, therefore, that of the total number (forty-six), thirty-two or 69.5 per cent. showed the presence of murmurs; and that of the fourteen under five years, eleven, or 78.5 per cent., had murmurs; while of the thirty-two between the ages of five and ten years, murmurs were present in twenty one, or 65.3 per cent.

Adding together the number of children examined consecutively in the preparation of our former paper and the forty-six examined consecutively in the preparation of this paper, we have a total of eighty of which number, fifty-three or 66.2 per cent. showed the murmur. Of these eighty children forty-eight were under five years of age, and of this number, thirty-two, or 66.2 per cent. had the murmur. Of the thirty-two remaining, all of whom were over five years of age, there were twenty-one, or 65.3 per cent., with the murmur. On the basis of these figures, therefore, it would seem that in institutional children, showing no special manifestations of disease, acute or chronic, but influenced only by their environment, between 60 and 70 per cent. have the type of murmur under discussion, and that this murmur is quite as common in children under five years of age as in later childhood. It is interesting to note also that these results correspond closely with those obtained by Rudolf, quoted above, and that the 31.6 per cent. of functional murmurs found by Butler in the examination of a series of hearts in children ranging in age from seven years to puberty would seem to support the suspicion suggested by our figures—that this murmur is more common during the first five years of life than between this age and puberty—a view which we have held for some years.

While this murmur is doubtless more common in children living in an environment such as obtains in institutions of the class we have worked in, we are, nevertheless, convinced from our experience in private practice among the better class of people that it occurs with almost equal frequency in children living under the best hygienic conditions, who have practically no evidences of anemia, and whose state of health is very slightly below the normal, and not infrequently in those who have every appearance of being perfectly well. Of course, they are much multiplied in frequency during the acute febrile conditions, or during any decided condition of ill health.

This murmur has no special clinical significance. The recognition of the frequency of its occurrence is important in order that it may be differentiated from murmurs of organic origin.

If its characteristics are fairly understood, it is not likely that it will be misinterpreted The points upon which one should lay special stress in differentiating it from organic murmurs are these:

The changes in the heart muscle found in organic disease of the heart, as indicated by displacement of the apex-beat and increase in the area of dulness, are not present. The murmur is proportionately more augmented by the recumbent posture than are the organic murmurs; it is often audible only when the patient is recumbent. It is differently influenced by respiration, being loudest at the end of expiration and the beginning of inspiration, sometimes entirely disappearing at the end of a full inspiration. The murmur invariably has its point of maximum intensity in the second left interspace close to the sternum or over the third costal cartilage. It is sometimes transmitted over practically the entire cardiac area, but rarely beyond it. It never eliminates the first sound, but exists only after the period of systolic closure; it is, in other words, late-systolic.

PAROXYSMAL IRREGULARITY OF THE HEART AND AURICULAR FIBRILLATION.

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The following case of marked irregularity of the heart occurred in the service of Dr. Peterson, professor of gynecology and obstetrics in the University of Michigan. Careful examination of the heart was made and a number of sphygmographic tracings were obtained. Unfortunately at that time we were not acquainted with Mackenzie's methods of taking the venous and liver pulses and failed to take advantage of this most valuable method of analyzing cardiac irregularity. The case is of considerable interest, however, and the light which seems to be thrown on it by our experience of irregularity in animal experiments encourages us to put it on record. The history of the case as supplied by Dr. Peterson is as follows:

Mrs. H. H., a widow, aged sixty-four years, was admitted to hospital, December 23, 1901, for ovarian fibroid. She was operated on December 24. Anesthesia (ether) for one hour and twenty minutes. Her condition was good at end of the operation; pulse 90. Nausea persisted throughout the 24th, and on 26th the patient vomiting repeatedly, but on the 27th some improvement was noted in this respect. The pulse on the 25th and 26th was 90 to 100 at the visiting hours, but the nurse stated subsequently that she had noted frequent irregularities.

On the 27th the pulse was 88[1] at 6 A.M.; 57 at 8; 53 at 10; 86 at noon; 51 at 2 P.M.; 92 at 4; 86 at 6; 52 at 8; 82 at 10; and 54 at midnight. At 2 P.M. the record states "pulse very irregular." Temperature 98° to 99°.

December 28. Slight nausea, and vomiting occasionally. Pulse weak and irregular. Sphygmograms taken in the afternoon indicate the same irregularities as occurred on the 29th. The attacks of irregularity did not last long. Strychnine $\frac{1}{30}$ grain, every four hours.

December 29, 8.30. Pulse very irregular, but regular at 9:15. In the afternoon pulse tracings were taken every hour, showing the following conditions: At 1.45 great arrhythmia; 2.45 pulse regular; 3.45 irregular again; 4.45 irregular and this persisted through the rest of the day.

On December 30 nausea and vomiting stopped, and subsequently there was general improvement. The periods of irregularity became less frequent and were confined almost entirely to the night. The patient says she wakes up with a start and the heart becomes very irregular at once.

On January 11, 1902, the heart was very irregular during the night, but afterward remained regular until January 18, when she was discharged from the hospital and reached home in the evening. The patient reported that her heart was very irregular during the following night.

The patient gives the following history of the trouble with her heart: She first noted the irregularity twenty years before, during an illness, and since then it has occurred whenever she was ill, excited, or exhausted. Vomiting, to which she is occasionally subject, has always been followed by heart irregularity. The tendency to irregularity seemed to increase about ten years before the operation, as she observed that less important occasions gave rise to it than previously. A letter received from the patient in 1905 states that for two years after the operation she suffered very little from her heart, but then some overexertion gave rise to cardiac irregularity again, and for the last year very slight exertion or exposure to extreme heat or cold has induced an attack.

The examination of the heart by the usual clinical methods during the stay of the patient in the hospital gave no clue to the cause of the irregularity. No enlargement of the cardiac dulness could be made out, there were no murmurs, and no objective signs of heart failure apart from the irregularity were present.

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[1] We attach no importance to these numbers as indicating the true rate of the heart, as it is quite impossible to determine the pulse-rate in the type of irregularity shown in the sphygmograms. The numbers given, however, indicate periods of irregularity at 8 A.M., 2 P.M., 8 P.M., and midnight, while the intervening counts indicate a return to the normal.

Analysis of the Sphygmograms. The pulse tracings were taken with Jacquet's sphygmochronograph, recording one-fifth seconds. The tracings obtained during the irregular periods were all of the same type and may be represented by some of those taken on December 29. The marker above the tracing records the time in one-fifth seconds, while the intervals between successive pulses are given below in hundredths of a second.

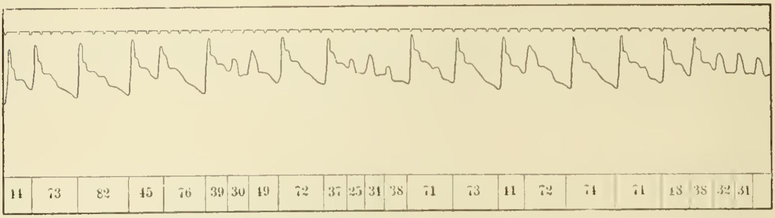

Fig. 1.—Pulse tracing at 1.45 p.m. December 29.

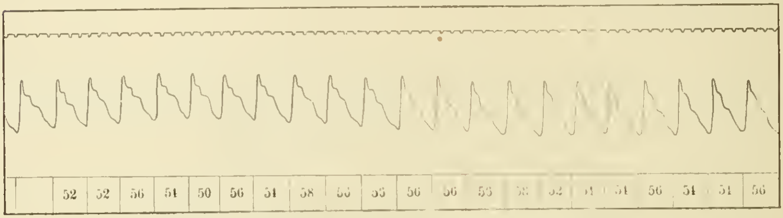

Fig. 2.—Pulse tracing at 2.45 p.m. December 29.

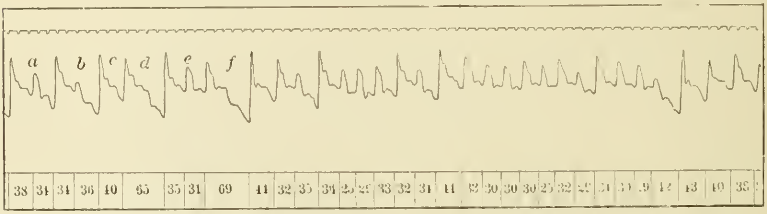

Fig. 3.—Pulse tracing at 3.45 p.m. December 29.

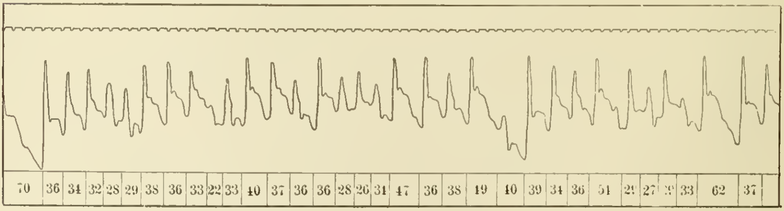

Fig. 4.—Pulse tracing at 4.45 p.m. December 29.

Of these four tracings, those taken at 1.45, 3.45, and 4.45 p.m. indicate marked and continuous irregularity, while the intervening one at 2.45 presents a quite satisfactory regular pulse of about 88 per minute. This rate is considerably above the average for the age of the patient, but is not to be considered as abnormal.[2] The slight variations in the intervals between the pulsations are not greater than those observed in normal persons. Auscultation at this time indicated no abnormality in the heart sounds; in short, the heart and circulation at this time appeared normal.

The tracings taken before and after this period of regularity offer a marked contrast, the pulse showing great variations in the strength of the beats. The general character of the tracings shows a number of large pulsations occurring at irregular intervals and having interspersed among them smaller pulsations, sometimes singly, at other times in twos or threes, or in groups of larger numbers.

Examination shows that the large excursions of the lever are not so simple as they appear at first sight. For example, in Fig. 1 the descent following the second and third rise of the lever is interrupted by oscillations which are obviously due to a beat of the pulse in each case too weak to make a more definite record. This is perhaps better shown in Fig. 3, in which at *a* and *b* the large excursion of the lever is followed by a weaker contraction, while at *d* the descent of the lever shows only a slight oscillation; yet there can be no question that there was here a secondary contraction similar in character to those at *a* and *b*, but so weak that it failed to cause even such a beat as is recorded at *b*. In the interval *c*, on the other hand, there was probably no interposed systole of the ventricle. When the stethoscope was applied over the heart there was found a duplication of the first sound in such intervals as have been recorded at *a*, *b*, and *d*, while there was no such duplication at *c*. At *e*, Fig. 3, two small elevations followed each other, and at *f*, a third one occurred which was too feeble to do more than arrest the fall of the lever for a moment. A little later, in Fig. 3, a succession of eleven rapid weak contractions are seen. Counting these weak and almost unrecorded contractions, the pulse is found to be beating in the irregular periods at 140 to 150 a minute. On auscultation the heart sounds were found to be very much more rapid than the pulse taken by the finger, which was given as about fifty a minute during the irregular periods.

Even when these small beats were taken into account the rhythm of the heart is very irregular; for example, during a succession of rapid contractions in Fig. 3, the interval between the successive pulses varies from 0.25 to 0.41 second. There exists no such "regular irregularity" as has been described under the terms pulsus bigeminus or trigeminus. In these, one normal pulsation is followed regularly by one or two movements of smaller dimensions, but in our tracings no such sequence is to be made out.

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[2] Mackenzie, The Study of the Pulse, p. 53.

Even more striking than the irregularity in the pulse rhythm in this case is the marked variation in the strength of the beats. This variation in strength is, of course, a very common phenomenon in irregularity of the heart. In the numerous tracings published of late years by Wenckebach, Mackenzie, Cushny, and others, this variation in strength is universally noted. But in most of these the strength of the beat varies with the length of the preceding interval; the ventricle accumulates more energy during a long interval and at the same time receives more blood from the auricle, and these two factors together cause a very powerful contraction and a very large output of blood. In this case, however, the size of the pulse bears no relation to the length of the preceding interval. It is true that when there has been a great fall in the pressure from the absence or inefficiency of the preceding beat, the next contraction is often very large; but this is obviously not the determining factor in most instances, for a strong contraction is often preceded by a shorter interval than a smaller one, and in other cases two intervals of equal length are followed by pulsations of very different height. The variations in the size of the pulse must be due, therefore, not to different degrees of recuperation in the contracting power of the ventricle, but rather to variations in the amount of blood which it receives from the auricle during its diastole. The amount of blood entering the ventricle may be altered by a variety of causes. One condition which usually causes pulses of different sizes is the beating of the ventricle and auricle at different rates. For example, if the auricle beats three times to the ventricle's twice, one of the auricular beats discharges no blood into the ventricle, because the auricle contracts against the contracted ventricle. The next pulse is correspondingly small. This condition, however, leads to a rhythmical variation in the strength of the pulse, as has been shown by one of us in the case of certain poisons which lead to a ventricular rhythm independent of, and different from, that of the auricle.[3] No such rhythmical variation in the size of the pulse is to be made out in this case, so that the condition is obviously different from that occurring in animals under the influence of these poisons. An irregular and intermittent beat of the auricle might, however, cause the occasional discharge of blood into the ventricle during the diastole and thus lead to a large pulse-wave from the next ventricular contraction; the ventricle's rhythm must have been much more rapid than that of the auricle, and, of course, must have been developed independently.

Another factor which may determine the variations in the strength of the pulse in this case is the imperfect diastole of the ventricle. The blood can flow from the auricle only when the intra-auricular pressure is higher than the intraventricular, and if the ventricle remains half-contracted during diastole, the inflow must be small and the next pulse correspondingly low. If this be the correct explanation of the variations in the size of the pulse, these are, of course, due to the ventricle alone. The auricle might be beating at the same rate as the ventricle, or it might be entirely inactive, the blood simply entering the ventricle from the auricle through the pressure exerted on it by the right heart.

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[3] Journal of Physiology, xxv, 49.

From the evidence of the tracing we are unable to determine which of these two factors determine the size of the pulse. If the venous pulse had been registered simultaneously with the arterial, much light would have been thrown on the point, and it would probably have been possible to decide whether slow, intermittent, irregular contractions of the auricle are the explanations, or variations in the degree of relaxation of the ventricle. In the absence of such evidence the latter appears the preferable. It is a feature familiar to everyone who has watched an irregular heart in the living animal, while it is extremely rare to find the auricle beating so much more slowly than the ventricle, as would be indicated by the number of full pulses.

The regular beat of the ventricle which is registered by the sphygmograph in normal persons follows an impulse which originates in the "rhythmic area" around the great veins of the heart and is propagated to the ventricle by way of the auricle. Two factors are thus involved directly: (1) The discharge of the impulse from the rhythmic area must be regular and its path through the auricle unobstructed; and (2) the ventricle must always be in such a condition that it can respond to the stimulus, while its irritability is too low to permit of its initiating a contraction spontaneously. Irregularity of the heart arises either from irregularity in the discharge of the impulses or from an abnormal condition of the ventricle, which leads to spontaneous contraction before the descending impulse reaches it; in rarer cases the ventricle is unable to respond to the descending impulse and a beat lapses. Careful examination of the pulse often determines whether the irregularity arises from ventricular disability or from the irregular action of the originating area.[4] In an intermittent pulse due to ventricular lesion the intermission is almost exactly twice the interval between two normal beats, while if the discharging area be at fault the intermission is considerably shorter. The fact that the intermission due to ventricular abnormality must be a multiple of the normal length of the pulse wave, follows directly from the consideration that the impulses descending through the auricle are undisturbed in rhythm, and the intermission can end only on one of these reaching the ventricle and originating the first normal beat after the irregularity. That the intermission is shorter when it is due to an irregular discharge from the rhythm-giving area was first deduced from animal experiments and has been found to conform with clinical experience in too many cases to permit of further doubt.

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[4] Cushny, Journal of Experimental Medicine, iv, 327; Mackenzie, loc. cit., p. 297; Wenckebach, Ztschr. f. klin. Med., 1899, xxxvi, 181, and Die Arythmie des Herzens, Leipzig, 1903.

In our case of irregularity there is no reason to suppose that the ventricle failed to respond to the impulses it received. The length of the apparent intermissions or of the periods of short and quick pulses is not a multiple of the single beats. For example, in Fig. 1 the first single beat is 0.44 second in duration, and if the next intermission had been due to the failure of the ventricle to contract on the next regular stimulus its length would have been 88 instead of 73. Similar reasoning in each case shows that the irregularity is due, not to variations in the ventricular receptivity of stimuli, but to an irregular discharge of impulses or to an irregular transmission through the auricle or auriculoventricular fibers.

The question arises whether these irregular impulses originate from the same point as those in the normal regular pulse, or whether the irregularity of the discharge is due to the fact that some other point takes up the function, and, being only imperfectly adapted to it, issues only irregular impulses. The latter view has been suggested by Mackenzie,[5] who holds that in certain forms of continued irregularity the ventricle assumes an automatic or endogenous rhythm, while the auricle may assume the ventricular rhythm or may remain quiescent or "paralyzed." Mackenzie supports this view by a careful analysis of his tracings and by excluding all other known factors; in the particular cases observed the interpretation may be correct, although the argument by exclusion of all known causes is dangerous and an alternative explanation might possibly suffice without the necessity of adopting the inversion of the cardiac rhythm postulated by him. But in animal experiments in which the ventricle has been induced to take up an endogenous rhythm by means of drugs no such irregularities of the heart rhythm are observed,[6] and in numerous unpublished experiments in which rapid induced shocks were applied to the ventricle, one of us was able to invert the cardiac rhythm, the ventricle giving the rhythm to the auricle, without rendering the rhythm of either irregular after the first few seconds. An endogenous ventricular rhythm may, therefore, be perfectly regular in the mammalian heart, and it cannot be assumed *per se* to be the explanation of irregularity in the tracings under discussion.

In our tracings, then, there is nothing to suggest that the ventricle originated the rhythm anywhere. Although we cannot exclude the possibility of the whole phenomenon being due to endogenous ventricular influence, it seems rather to have merely responded to the impulses which it received at irregular intervals from the auricle. Not infrequently the impulses followed each other so rapidly that the ventricle could not dilate sufficiently in the interval to permit of the entrance of blood from the auricle. In other cases blood failed to enter in quantity, although there seemed to have been sufficient interval, and in this case the ventricular contraction caused a very small pulse wave.

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[5] Study of the Pulse, Brit. Med. Jour., March 5, 1904.

[6] Cushny, Jour. of Physiology, xxv, 49.

The conditions of the auricle and rhythmical area in the irregular periods appear to have been one of extreme activity, if, as we believe, the rhythm in this case was given to the ventricle by some other part of the heart. This impulse-emitting area was obviously in a state of great irritability, for the impulses amounted to 150 a minute, and at the same time the rhythm was extremely irregular.

The sudden onset of irregularity and its temporary character suggest that while there may be cardiac conditions favoring irregularity, the actual change from the regular beat must be due to affection of other organs. And the fact that it accompanied the nausea and vomiting and tended to occur when no exertion had been made, and especially in the night-time, suggest that the immediate cause of the irregularity was some abnormality of the central nervous system acting on a damaged heart. We are aware that the tendency at present is to regard such conditions as of peripheral origin and to exempt the central nervous system from responsibility in such phenomena. At the same time, the history of the case inevitably suggests this view, and we hope in the following pages to show indisputably that the central nervous system is capable of giving rise to very marked abnormalities of the heart action.

ANIMAL EXPERIMENTS.

In the course of the long series of experiments and demonstrations on the dog's heart carried out in the pharmacological laboratory of the University of Michigan, during the last ten years, it has happened occasionally that on opening the chest the heart was found to be beating very rapidly and irregularly. Doubtless other workers in this field may have had similar unfortunate experiences, but we are unaware of any recorded cases. Our dogs were anesthetized with morphine, 0.2 to 0.3 gram hypodermically, and chloretone administered by the stomach tube. The operation consisted in performing tracheotomy, prolonging the median incision to the lower end of the sternum, sawing through the sternum along its whole length, and hooking the two sides of the chest apart, thus exposing the pericardium, which was opened. The myocardiograph was then applied to the ventricle and auricle and tracings taken on a kymograph with smoked paper. The anesthesia was invariably deep enough to prevent any manifestation of pain and the eyelid reflex was absent before the incision was made. Sometimes, however, spontaneous respiratory movements returned while the sternum was being cut through, or if these were present before they became quickened and deepened. It was soon noted that when this change occurred, the tendency to cardiac irregularity was much greater than in those experiments in which more profound anesthesia had been induced. Attempts were made to record the changes in the pulse during the operation, but in the experiments in which this was done the irregularity was not developed. In several cases, however, it was noted that the heart rhythm was normal before and during the first part of the operation, but that when respiratory movements were induced by cutting through the sternum, the pulse, which had previously been of the usual slow vagus type seen in the dog, suddenly became accelerated and irregular. On examination of the heart in these cases, before the apparatus was applied to it, the ventricles were found in rapid irregular movement; the relaxation was often very imperfect between three or four successive contractions and then more complete for one or two beats. The impression was given that the ventricles were responding to a series of very rapid impulses which prevented their diastole, and that they could only relax when their irritability was reduced by fatigue, and then the more complete diastole followed. The auricles were widely dilated and no systole occurred in them; they were not wholly paralyzed and inactive, however, for on close inspection the fibers proved to be in a state of continual inco-ordinated contraction, each part of the auricle undergoing continuous fibrillary contraction independent of all other parts. The heart was in fact in the condition known to physiologists as auricular delirium, or fibrillary contraction in the auricle.

Auricular delirium is not infrequently produced by poisonous doses of bodies of the digitalis series,[7] and is very readily induced by rapid induction shocks passed through the auricular walls in animals. Several papers[8] dealing with the subject have appeared recently from the laboratories of Berne and Brussels, and we have also devoted some attention to it. In this condition the ventricle beats much more rapidly than normally and the beats are irregular in rhythm and vary in the degree of systole and diastole. Stimulation of the vagus, in our experience, which coincides with that of Kronecker and Spallitta, has no effect whatever on the movements of the auricle, while the ventricle is rendered slower and more regular or may be arrested completely. Phillips believes that the fibrillation of the auricle is lessened by stimulation of the vagus. It may be mentioned that the tremor in the tracing of the auricle during delirium often appears less marked, but this is due to the fact that the ventricle is slower and communicates less movement to the auricle, the fibrillation of the auricle causing no movement of the lever in itself. The more regular contraction of the ventricle under vagus stimulation is obviously due to the fact that the impulses from the auricle pass less rapidly through the block; when they are altogether excluded by section of the auriculoventricular band the ventricle beats regularly (Fredericq). The pulse, as well as the movements of the ventricle and auricle, were recorded by Phillips; during the auricular fibrillation the pulse was accelerated and very irregular, as we have also observed in our experiments.

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[7] Cushny, Journal of Exp. Med., ii, 233.

[8] Kronecker and Spallitta, Arch. Internat. de physiologie, ii, p 223; Philips, ibid., ii, p. 271; Fredericq, ibid., ii. p. 281.

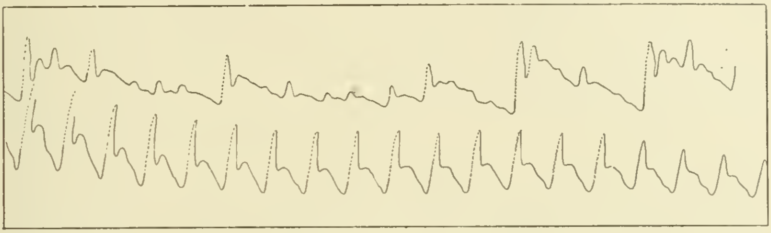

Fig. 5.—Pulse tracing from dog's carotid taken with Hurthle's tonometer. The lower tracing is normal; the upper was taken during auricular fibrillation from rapid electrical stimulation of the auricle.

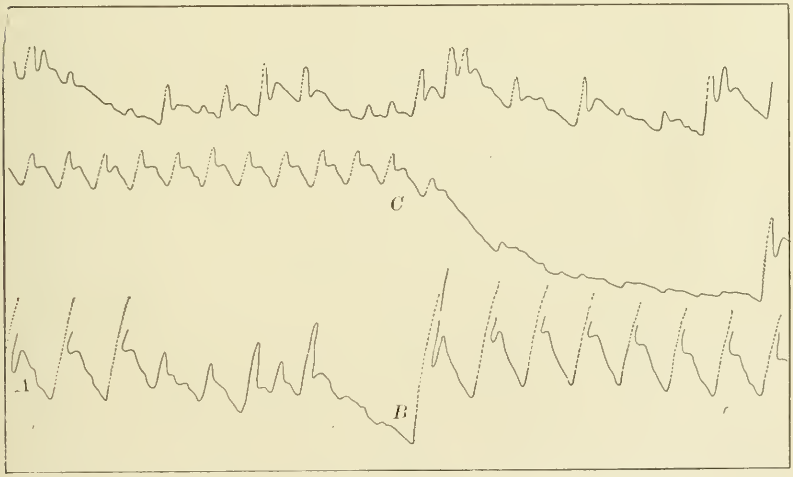

Fig. 6.—Pulse tracing from dog's carotid taken during auricular fibrillation, as in Fig. 5. At *A* feeble electrical stimulation of the auricle caused slight irregularity; *B*, stimulation discontinued and heart returned to normal; *C*, the heart previously normal received rapid electrical shocks in the right auricle which began to fibrillate; the uppermost tracing was obtained during auricular fibrillation.

Stimulation of the dog's auricle with rapid induced shocks causes fibrillary contractions which pass off when the current is shut off or very soon afterward. On repetition several times, the interval between the cessation of stimulation and the return of the normal auricular contractions becomes longer until finally it may continue for an hour or more after the electrodes have been removed. Such cardiac depressants as chloroform have no remedial effect even when pushed until the ventricle almost ceases under them. The only way in which we have been able to restore the auricle to regular contraction in persistent fibrillation, and this is not successful except occasionally, is by the application of cold directly to the heart. Large quantities of chilled salt solution poured on the exposed heart sometimes restored it to regular contraction.

The auricular delirium, which we have described as occurring in dogs during the operation, was obviously the result of sensory stimuli carried from the chest wall to the central nervous system. This might affect the heart either by producing changes in the circulation (shock) or by nervous impulses passing along the cardiac nerves to the heart. In the descriptions of shock given in surgical textbooks, the heart is often said to be irregular, but personal inquiry among our surgical friends failed to elicit any information in regard to the character of the irregularities, and we were led to the conclusion that the cardiac irregularity in shock is extremely rare or only seen in moribund cases. We are, therefore, disposed to attribute the condition to reflex nervous influences. These impulses must reach the heart either by the vagus or by the sympathetic fibers. And as a matter of fact, we have observed one instance in which vagus stimulation was followed by delirium. In this dog the heart was peculiarly irritable and much accelerated. The myocardiograph had been attached to the right ventricle and auricle and a tracing was being taken. The effects of electrical stimulation of one vagus in the neck were being demonstrated and the usual response followed, arrest of the auricle and the ventricle in diastole. But when the current was shut off, the heart, instead of returning to its normal rate, passed into fibrillary contractions in both auricle and ventricle. This is the only instance in which we have seen the unpoisoned heart affected in this way from the cessation of inhibition. In several experiments in which the irritability of the heart was exaggerated by large doses of the members of the digitalis series or of barium, section of the vagus led immediately to fibrillary contractions in the auricle and ventricle.

As has been stated, we have obtained no tracings of the pulse in dogs in which the heart passed into auricular delirium during the operations. The phenomenon is a rare one in our experience, although we have observed it sufficiently often to have no doubt as to its occurrence and as to its association with the operation. In several experiments the pulse was recorded during the operation by means of von Frey's tonometer attached to the carotid artery and the anesthesia was maintained at a point at which, while pain was absent, the reflexes were retained as far as possible. In none of these, however, was the auricular delirium developed, although the pulse was much accelerated; occasionally intermissions of the pulse occurred of the auricular type, indicating perhaps a tendency to fibrillation. The acceleration was very slight or entirely absent in experiments in which atropine had been previously injected in quantities sufficient to paralyze the cardiac vagus. The acceleration in dogs without atropine was due, therefore, for the most part to lessened vagus tone from the centre, being inhibited by impulses from the seat of the operation. It seems likely that the auricular delirium observed in rare cases in dogs during the operation is similarly to be explained by the disappearance of inhibition; the auricles in these cases, however, must be in some quite unusual condition of irritability.

Of course, we cannot claim to have shown any connection between this type of irregularity in the dog's heart and that in the case described. At the same time there exists similarities between them, and the sudden arrest of the irregularity in each suggests a common cause; in the dog the site of the lesion is the central nervous system, and the history of the patient suggests that here also the irregularity was of central origin. We had hoped to have the opportunity of continuing the investigation in other cases of irregular heart, but as circumstances preclude our working further together, we have decided to put the imperfect investigation on record, in the hope that others may be interested in the suggestion made and may be able to prove or disprove its correctness.

SUMMARY. A case of paroxysmal arrhythmia, with marked acceleration of the heart, is described, and it is shown that the irregularity is due to irregular discharge of impulses and not to defects in the contraction of the ventricle which appears to respond to the impulses received. A similar form of irregularity in the dog's heart is described, and it is shown that this is due to the ventricles receiving irregular stimuli from the auricle which is in the state of fibrillation. This form of arrhythmia is shown to occur in the dogs occasionally from peripheral irritation and it is probable that this gives rise to reflex inhibition of the vagus centre which, acting on an abnormal heart, causes auricular fibrillation. The suggestion is made that in the patient described and in other cases of paroxysmal arrhythmia the condition is due to auricular fibrillation from inhibition of the vagus centre.

HEMOCHROMATOSIS WITH DIABETES MELLITUS.[1]

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The clinical and pathological features of hemochromatosis were presented before this Association in 1899 by Dr. Opie,[2] who reported a fatal case of the disease. Opie's paper gives a complete bibliography of the subject up to the date of publication. At the same meeting, Dr. Osler exhibited a patient who showed some of the leading characteristics of the affection. This case was[3] later published in detail.

There are three cardinal symptoms manifested by hemochromatosis in its most advanced stages: (1) Pigmentation of the viscera, and usually also of the skin; (2) cirrhosis of the liver, almost invariably of the hypertrophic type and accompanied by striking pigmentation of the organ; and (3) diabetes mellitus, which dominates the clinical picture in the latest stage of the disease. The latter is due to the final development of a pigmentary cirrhosis of the pancreas, thus interfering with its glycolytic function.

In Opie's and Osler's cases the disease had not reached the diabetic stage. Death in Opie's case, was precipitated by an attack of typhoid fever. Osler's patient presented a striking pigmentation and had a very large liver. Efforts have been made to trace the course of the disease in the latter case, but the patient has been lost from view. I have the privilege[4] of reporting herewith two fatal cases of hemochromatosis from the medical wards of the Johns Hopkins Hospital, in which diabetes was present and was responsible for the death of the patients. The paper deals only with those cases of hemochromatosis in which the diabetic stage has been reached. In 1899 Anschütz,[5] including one of his own, collected twenty-four cases of hemochromatosis with diabetes. In a review of the literature since that date I have been able to find nine additional cases, which, with the two here reported, make a total of eleven cases reported since 1899. Altogether, therefore, thirty-five cases of hemochromatosis with diabetes have been recorded up to the present time.

Hemochromatosis is a rare disease. In the first seventeen years since the opening of the Johns Hopkins Hospital up to May 15,

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[1] Read at the meeting of the Association of American Physicians, Washington, D. C., May 15 and 16, 1906.

[2] Transactions of the Association of American Physicians, 1899, vol. xiv, p. 253. (For the literature previous to 1899 see the bibliography as given by Anschütz and Opie.)

[3] Brit. Med. Jour., 1899, vol. ii, p. 1597.

[4] I am indebted to Dr. L. F. Barker and Dr. W. S. Thayer for the opportunity of reporting these two cases.

[5] Deut. Arch. f. klin. Med., vol. lxii, p. 411.

1906, there have been only three cases in the medical wards and dispensary, in which, from the clinical features and the finding of hemosiderin in the skin, the diagnosis was without doubt. These cases include Osler's case and the two here reported out of a total of 106,000 medical cases treated in the wards and dispensary. As already stated, only two of these had reached the diabetic stage. That hemochromatosis is an uncommon cause of diabetes is indicated by the fact that it was responsible for the disease in only two instances out of a total of 256 cases of diabetes treated in the medical wards and medical dispensary up to May 15, 1906. The histories of these two cases follow:

Case I. (*Gen. Hosp. No.* 46430).—*Moderate pigmentation of the face, neck, and hands. Iron demonstrated in the epithelial cells of the sweat glands. Very large liver. Time of onset of pigmentation uncertain. Death from coma twenty months after the onset of the diabetic symptoms.*

E. W. S., male, white, married, aged forty-five years, cigar manufacturer, a resident of Virginia, was admitted to the Johns Hopkins Hospital, April 21, 1904, complaining of "diabetes."

Family History. The only feature of interest was that one brother had died of diabetes at the age of forty-nine.

Personal History. The patient had measles and whooping-cough as a child. Since then the only serious illness was pneumonia at thirty-three. He had gonorrhœa at twenty-five. No lues. Smoked six to ten cigars a day. Previous to two years before admission he had been a heavy drinker, taking from ten to thirty drinks of whiskey daily. His average weight when he was well was 195 pounds.

Present Illness. In January, 1903, the patient began to have marked polyuria and thirst. This was accompanied by increasing weakness and emaciation. He had lost sixty-five pounds since January, 1904. His appetite was ravenous. For three months he had suffered from a general pruritus. Recently a papulosquamous eruption had appeared on the legs and thighs. During the last two months there had been severe cramps in the muscles of the legs. The sexual power had been markedly impaired for a year. Neither the patient nor his friends had been struck by any change in his color.

Physical Examination. The patient was of medium stature and still fairly well nourished. A feature that attracted one's attention at once was a definite but not extremely marked, slaty-brown pigmentation of the face, neck, and hands. The only other situation in which the pigmentation was increased was about the scrotum. In front of the right external malleolus there were three fading purpuric spots. There was no pigmentation of the sclerotics nor of the buccal mucous membrane. No jaundice.

The radial vessels were slightly thickened. The blood-pressure was 123 mm. Hg. The pulse was otherwise normal. The breath possessed a faint, sweetish fruity odor.

The lungs showed signs of slight emphysema. The heart was normal.

The liver was markedly enlarged. The relative hepatic dulness commenced at the middle of the fourth interspace in the mammillary line. The absolute hepatic dulness began at the middle of the sixth rib and extended to a point in the mammillary line 5 cm. below the costal margin and in the median line to a point 14 cm. below the tip of the ensiform cartilage and only 3.5 cm. above the umbilicus. The left lobe of the liver was even more markedly enlarged, its edge disappearing beneath the left costal margin in the anterior axillary line. The edge of the liver was rounded and everywhere well felt. The liver surface was smooth.

The border of the spleen could not be felt. It appeared to be enlarged, however, as its percussion measurements were 10 x 22 cm.

The abdomen revealed a distinct fulness corresponding to the enlarged liver. In the epigastrium there was a properitoneal hernia. There was nothing otherwise to be made out on abdominal palpation. Ascites did not exist.

The deep reflexes were markedly diminished but were still to be elicited. The superficial reflexes were normal.

The ophthalmoscopic examination made by Dr. Mills showed no changes in the fundi.

Blood Count. Red cells 4,800,000 per c.mm.; leukocytes, 7700 per c.mm.; hemoglobin 87 per cent. An examination of a fresh specimen failed to show any evidences of lipemia.

Frequent examination of the stools revealed no excess of fat. Toward the latter part of his stay in the hospital, the patient had an annoying diarrhœa, having from five to ten loose movements daily.

Histological Examination of the Skin. Sections were made by Dr. Opie of a piece of pigmented skin removed from the back of one of the hands, and a marked reaction for iron was found in the epithelial cells of the sweat glands by the potassium ferrocyanide and hydrochloric acid method.

Summary of Urine Examinations. The total amount of urine for the first full twenty-four hours (April 22 to April 23) was 3200 c.c. The urine was clear and of a very pale yellow color. Acid reaction; specific gravity, 1033. No albumin. Sugar, 7 per cent., or 224 grams in the twenty-four hours. There were definite reactions for acetone and diacetic acid, although they were not intense. The microscopic examination was negative.

The patient was put on a non-carbohydrate diet by gradual withdrawal of the starches. After being on a strict diet for nine days he failed to become aglycosuric and was still excreting on the ninth day 75 grams of glucose. As he was losing weight, moderate amounts of starch were allowed. The reactions for acetone and diacetic acid remained positive throughout the patient's entire stay in the hospital.

On May 12, the day before he left the hospital, he voided 1800 c.c. of urine. It was acid; specific gravity 1029; no albumin; sugar, 3.1 per cent. or 55.8 grams; acetone and diacetic acid positive; no casts.

The patient was discharged on May 13, 1904. His condition had not materially changed. The pigmentation had apparently remained about the same. The enlargement of the liver persisted. A clinical diagnosis of hemochromatosis and diabetes mellitus was made.

Dr. J. H. Neff, of Harrisonburg, Va., who had charge of the patient after he returned home, informed me that during the summer a superficial ulcer developed on one of the legs. This slowly healed. There was no special increase in the degree of pigmentation. The patient's condition gradually became worse and he died in coma on September 6, 1904, twenty months after the onset of his diabetic symptoms. Unfortunately no autopsy was obtained.

CASE II. (*Gen. Hosp. Nos.* 52486, *and* 53388).—*Intense pigmentation of many years' standing. Iron demonstrated in the cells of the sweat glands. Enormous liver. Symptoms of diabetes for six months before death. Death from bronchopneumonia and not from coma.*

J. W. S., male, white, married, bricklayer, aged fifty-three years, was admitted to the Johns Hopkins Hospital for the first time on October 6, 1905, complaining of weakness, thirst, and frequent micturition.

Family History. There was nothing of importance bearing on the patient's case.

Personal History. The patient had always been a very healthy man with the exception of an attack of pneumonia seventeen years ago. He denied gonorrhœa and syphilis. He had smoked and chewed tobacco in moderation. Alcohol was used very moderately, three glasses of beer a week. He was the father of eight children. His average weight was 160 pounds.

Present Illness. Six weeks before admission the patient first noticed that he was voiding urine more frequently and in larger amounts, and that he had persistent thirst. These symptoms were preceded for a short time by increasing weakness. During the last four weeks he had lost twenty pounds. His appetite had not been voracious. The bowels were constipated. Six days before admission he had to give up work owing to the increasing physical weakness. For three days he had noticed a diffuse, red eruption over the lower part of the chest, the abdomen, and genitals.

The patient had not noticed any recent change in his color. His daughter said that his skin had been dark for many years and as long as she could remember.

Physical Examination. The patient was fairly well nourished, his weight on admission being 132 pounds. The mucous membranes were of good color. One was immediately struck by the remarkable pigmentation of the skin. This was most marked over the face, neck, and hands, where the skin had a very dark-brown color. The skin of the whole body was much more pigmented than normal, and there was intensification of the color in the axillæ. Over both axillary regions, the lumbar regions of the abdomen, and the groins there was a bright erythema which disappeared on pressure. There was no pigmentation of the sclerotics nor of the buccal mucous membrane. Below the left eye there was a xanthoma plaque measuring about 3 mm. in diameter.

The radial vessels were moderately sclerosed.

The examination of the heart and lungs revealed nothing abnormal.

The liver was enormously enlarged. The relative hepatic dulness commenced at the level of the fourth rib in the mammillary line. The absolute hepatic dulness began at the fifth rib and extended to a point 12.5 cm. below the costal margin in the mammillary line, which was 2 cm. below the level of the umbilicus. In the median line the absolute dulness reached just to the level of the umbilicus. The left lobe was markedly enlarged. The edge of the liver was everywhere readily felt and somewhat rounded. The surface was smooth. The edge of the spleen was easily palpable at the costal margin on deep inspiration. The examination of the abdomen was otherwise negative. There was no ascites.

Both knee-jerks were present but slightly diminished.

Blood Examination. Red cells, 5,304,000 per c.mm.; leukocytes, 9200 per c.mm. Hemoglobin, 95 per cent. (Gowers). The differential count showed no special variations from the normal. There were no evidences of lipemia by either chemical or microscopic tests. On October 9, Dr. Walter Jones examined the blood spectroscopically and reported that only the normal bands of oxyhėmoglobin were present.

Microscopic Examination of the Skin. On October 21, a small portion of the skin from the left shoulder was excised to be examined microscopically for iron. The examination was made by Dr. Bunting, who reported as follows: In the Malpighian layer of the epidermis the cells show an excess of fine pigment granules which do not take the Prussian-blue color with the ferrocyanide test. In some of the epithelial cells of the sweat glands and ducts, however, there are present coarser, more refractile granules, which do stain a brilliant greenish-blue and which are granules of hemosiderin. There were no large vessels found giving smooth muscle in which to look for hemofuscin.

Summary of Urine Examinations. For the first full twenty-four hours (October 7 to October 8) 1240 c.c. of urine was voided. It was of amber yellow color; acid; 1034; no albumin; sugar, 4.8 per cent., or 59.5 grams in twenty-four hours; acetone and diacetic acid were present; no casts.

Throughout the patient's stay in the hospital sugar was constantly present, the largest excretion being 272 grams during the twenty-four hours on a mixed diet. The lowest excretion of sugar on a strict non-carbohydrate diet was 39 grams. He failed to become aglycosuric on October 21, when a "hunger-day" was instituted, 23 grams of sugar being excreted. Acetone and diacetic acid persisted in the urine throughout his entire stay in the hospital.

In order to see whether the urine gave any indications of excessive blood destruction it was tested, on October 19, for hematoporphyrin, but the spectroscope failed to give the characteristic absorption bands.

On October 21 the patient was discharged. His general condition had materially improved during his stay in the hospital, and he gained one pound in weight.

Second Admission. The patient was readmitted to the hospital on January 9, 1906. His main symptoms in the interval were a "tired, sleepy feeling," great weakness across the small of the back, and some swelling of the ankles.

The examination of the liver, skin, and urine revealed practically the same features as on the first admission. The urine contained from 4 to 6 per cent. of sugar, and there were quite marked reactions for acetone and diacetic acid. He was discharged on January 13, feeling about the same as on admission.

Observation Outside the Hospital. After leaving the hospital, I was able to follow the course of the disease. The patient progressively failed. His main complaint was drowsiness and great weakness in the lumbar region. His disposition changed and he would at times have violent fits of anger. The reactions for acetone and diacetic acid became very intense and persisted for a long time, even with large doses of sodium bicarbonate. Small amounts of carbohydrates were permitted daily. During the night of February 11, he had a chill and the temperature rose to 100.2° F. The next day patches of bronchopneumonia were detected over the lower lobe of the left lung. Fearing the onset of coma the sodium bicarbonate was increased to 4 grams every two hours and the patient was put on a diet of eggs, with a quart of milk and a half-pint of cream daily.

On February 14, contrary to expectation, the reactions for acetone and diacetic acid had entirely disappeared. There was 2.4 per cent. of sugar present. The physical signs of bronchopneumonia were apparent. The pulse was 132 and much weaker. The respirations were 32 to the minute, but had no suggestion of an "air-hunger" type. Although the patient was dull this seemed to be due rather to his general weakness than to advancing coma. He died suddenly at 12.10 A.M. on February 15. Every effort was made to secure an autopsy, but the patient's family refused permission.

It was most unfortunate that autopsies could not be obtained in these two cases. Both patients died after leaving the hospital. I fully appreciate the risk in making a diagnosis of hemochromatosis without the evidences afforded by an autopsy. Personally I am fully convinced that both were cases of diabetes due to hemochromatosis. Each possessed the triad of symptoms characteristic of hemochromatosis in its final stage: pigmentation of the skin, hypertrophic cirrhosis of the liver, and diabetes of a severe type. Even with this symptom-complex one would hesitate to make a diagnosis of hemochromatosis without the finding of iron in the skin. In both these cases, however, hemosiderin was demonstrated in the epithelial cells of the sweat glands, in the first case by Dr. Opie, and in the second case by Dr. Bunting. I have, therefore, little hesitation in placing these two cases on record as instances of diabetes due to hemochromatosis.

In neither case was there any demonstrable evidence of blood destruction. In each the blood count was practically normal. In Case II the blood was examined spectroscopically for abnormal absorption bands, and the urine by the same means for hematoporphyrin with negative results. The only evidence of possible blood or vascular changes was in Case I, in the existence of a few small purpuric spots about one of the ankles. In Case I it was impossible to determine the relationship of the time of the onset of the skin pigmentation to the liver changes and diabetes. In Case II, however, the pigmentation undoubtedly antedated the diabetic symptoms which had existed for only six months before death. The skin pigmentation, having existed for as long as members of his family could remember, presumably also antedated the hypertrophic cirrhosis of the liver.

Before preceding to a brief consideration of the pathogenesis of hemochromatosis, I shall give a very brief summary of the cases of this disease associated with diabetes mellitus that have been reported since the appearance of the paper by Anschütz in 1899. The names of the observers and a brief synopsis of the cases follow:

I. Henry W. Berg.[6] Female; aged forty-four years; pigmented skin; exophthalmic goitre; enlarged liver; diabetes. Patient alive when reported. Skin not examined for iron.

II. A. Murri.[7] Female, aged fifty-nine years; pigmentation of arms; pediculi present; liver and spleen not enlarged. Highest percentage of sugar 4.3. Patient discharged "well." Skin not examined for iron.

III. K. Ubert.[8] Male; physician; age not given; 7 per cent. of sugar; enlarged liver and spleen. Death from coma. Diagnosis confirmed at autopsy.

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[6] Med. Record, 1899, vol. lvi, p. 881.

[7] Wiener klinische Rundschau, vol. xv, p. 345.

[8] Dissertation. Ueber Hemochromatolyse mit Pankreasatrophic und Diabetes (Zurich) Affoltern, 1902, J. Weiss.

IV. R. Lepine.[9] Male; aged forty-five years; 71 grams sugar in twenty-four hours; diabetes lasted eight months; enlarged liver and spleen; diagnosis confirmed at autopsy.

V. J. W Beattie.[10] Male; aged thirty-nine years; pigmentation of the skin, which was thought to have followed the diabetes; death from coma; pigmentary cirrhosis of the liver and pancreas found at autopsy.

VI. L. Margain.[11] Male; aged forty-seven years; pigmentation of skin; relationship of onset of pigmentation to onset of diabetes not determined; enlarged liver. Autopsy.

VII. Hess and Zurhelle.[12] Case I. Male; aged fifty-two years; pigmentation of skin; diabetes; autopsy, showing hypertrophic pigmentary cirrhosis of the liver and sclerosis of the pancreas with pigmentation.

VIII. Hess and Zurhelle.[13] Case II. Male; aged fifty-three years; marked alcoholic; pigmented skin; severe diabetes; autopsy, showing hypertrophic cirrhosis of the liver and a pigmented, firm pancreas.

IX. G. H. Moll van Charante.[14] Male; aged thirty-two years; skin pigmentation; enlarged liver; severe diabetes; death from coma; autopsy, showing general hemochromatosis.

In 1882, Hanot and Chauffard first described two cases of diabetes mellitus associated with hypertrophic cirrhosis of the liver and a bronze-like pigmentation of the skin. Four years later Lettulle reported two similar cases. In 1886, Hanot and Schachmann reported a fifth case. As all these cases agreed in that there was diabetes, a pigmentary hypertrophic cirrhosis of the liver, and, with the exception of Hanot and Schachmann's case, pigmentation also of the skin, the latter clinicians felt that they had established the existence of a new form of cirrhosis, *cirrhose pigmentaire diabètique*, as well as a new form of diabetes, *diabète bronzé*.

The majority of French clinicians have held that the diabetes is primary; that the diabetic alteration of the blood causes a destruction of the red cells, with the deposition of the blood pigment in the viscera and in the skin, giving rise to the so-called bronzed diabetes.

When von Recklinghausen, in 1899, described a general pigmentation of the viscera under the name of "hemochromatosis," it became at once apparent that this condition was closely related to bronzed diabetes. von Recklinghausen found that the tissues in hemochromatosis contained two varieties of pigment. One of these, "hemosiderin," contains iron, and possesses a brown color, and is thought to be derived from the hemoglobin of the blood. It is found in the cells of the lymphatic glands, liver, pancreas, and the sweat glands. The other, "hemofuscin," contains no iron, is finer, of an ochre-yellow color, and is present in the smooth muscle cells of the stomach, intestines, bloodvessels, and lymphvessels, but rarely in those of the urinary bladder, ureter, and vas deferens. It is also found in the connective-tissue cells of certain localities—for example, Glisson's capsule, the trabeculæ of the spleen, and the sheaths of the bloodvessels.

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[9] Lyon médical, 1903, vol. c, pp. 593 and 652.

[10] Journ. of Path. and Bact., 1903–1904, vol. ix. p. 117.

[11] Revue de Médecine, 1905, vol. xxv, p. 214.

[12] Zeitschrift f. klinische Medizin, 1905, vol. lvii, p. 344.

[13] Ibid.

[14] Neiderl. Tijdschr. v. Geneeskunde, Amst., 1905, vol. xli, p. 1727.

As more cases of hemochromatosis were studied, the view gained ground that in this condition some unknown agent caused an increased vulnerability of the red cells with dissolution of the hemoglobin and deposition of the two pigments in the situations already noted. The deposition of the hemosiderin in the cells of the liver and pancreas and of the hemofuscin in the connective tissue of these glands eventually leads to the large, cirrhotic, pigmented liver and the sclerotic pigmented pancreas which are so characteristic of the disease. The general belief now is that diabetes develops in hemochromatosis only when the pancreatic tissue becomes so seriously diseased that the islands of Langerhans are largely destroyed. The glycolytic ferment, which is supposed to be formed in these islands, is no longer produced and is not able to render active, according to Cohnheim, another ferment-like body produced in the muscles, a function which, according to this observer, is necessary for the proper utilization of the carbohydrates. Consequently, an accumulation of glucose results, a hyperglycemia occurs, and permanent diabetes ensues. The diabetes is of a severe grade and dominates the picture in the late stages of the disease. Acidosis is nearly always present and the majority of patients die of diabetic coma. Death usually occurs within one year after the onset of the diabetic symptoms, although it was deferred for twenty months in the first case here reported.

As to the relationship of the diabetes to the pigmentation the above view is the one generally held. The pigmentation of the viscera is primary and antedates the diabetes. The skin is not always pigmented in hemochromatosis. There may be visceral pigmentation long before there is any bronzing of the skin. The view formerly strongly supported by the French clinicians that the diabetes is primary and responsible for the pigmentation and visceral changes has now few adherents.

The basal cause of hemochromatosis still remains an unsolved problem. The view generally held is that some unknown toxic agent causes an increased hemocytolysis of the red cells. In a few of the reported cases there has been some evidence to support this view, such as the rare occurrence of purpura and the existence of hemoglobinuria in Case II of Hess and Zurhelle. On the other hand, Meltzer[15] has suggested that the absence of any anemia in these cases is strongly against the view that there is increased blood destruction in hemochromatosis. In a discussion of Berg's case he cites the fact that there is an absence of general hemochromatosis in pernicious anemia, in which there is increased blood destruction, as an argument against the view that hemochromatosis is due to a pathological condition of the red cells. He is inclined to think that the excess of iron in the tissues is due to a disturbance of the mechanism of metabolism which has to do with the removal of iron from the system. In other words, there may be an iron retention. As the amount of iron eliminated in the feces about equals that absorbed by the system from the food, he thinks that some light may be thrown on the subject by a quantitative analysis of the iron excreted in the stools.

The most thorough pathological and chemical study of hemochromatosis in recent years is that by Hess and Zurhelle. They strongly support the view that the cirrhosis of the liver and the pigmentation of the tissues are dependent on some common cause. They hold that some toxic substance, possibly alcohol, causes disturbances of metabolism which bring about both of the above changes. A lipemia, which was present in one of their cases, is explained in the same way. Their investigations also go to show that a sharp distinction between hemosiderin and hemofuscin cannot be made. They claim to have found them side by side in the same cell with gradual transitions from one to the other.

Of the thirty-five recorded cases of bronzed diabetes, including the two here reported, all but two were in males. These two have been reported since Anschütz's analysis of twenty-four cases in 1899, at which date he found that all the reported cases were in males. Cases in women have been reported by Berg, of New York, and Murri, of Bologno. The records of these cases, however, leave a doubt in one's mind as to whether they were true cases of bronzed diabetes. Neither came to autopsy and there is no report of iron having been examined for in the skin. It may be mentioned here that a case of hemochromatosis in a female was reported by Dr. Maude Abbott. As this patient did not have diabetes it has not been included in this series. The ages of the patients ranged from thirty to sixty years, with the exception of one patient who was sixty-one. The diabetes, as already stated, is the final stage of the disease and is in no special sense different from other cases of this affection. It is of a severe form and almost invariably causes death from coma within one year after its onset.

Summary. Hemochromatosis with diabetes mellitus (bronzed diabetes) is a rare disease. Of 256 cases of diabetes, out of a total of 106,000 medical admissions to the wards and dispensary of the Johns Hopkins Hospital, two were due to hemochromatosis. Of thirty-five cases in the literature, including the two here reported, all but two were in males. With one exception the ages of the patient ranged from thirty to sixty years.

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[15] Medical Record, 1900, vol. lvii, p. 43.

Hemochromatosis in its final stages presents a triad of symptoms: (1) Pigmentation of the viscera and, in the vast majority of cases, also of the skin; (2) hypertrophic cirrhosis of the liver—very occasionally atrophic cirrhosis; and (3) diabetes mellitus, a terminal feature, due to a sclerosis of the pancreas causing destruction of the islands of Langerhans and consequent interference with the secretion of the normal glycolytic ferment.

Death usually occurs within a year after the onset of the diabetic symptoms, although it was delayed till twenty months in Case I, herewith reported.

The balance of opinion is in favor of the view that hemochromatosis is due to some toxic agent of unknown character and origin, which results in an increased destruction of the red blood cells with deposition of hemosiderin and hemofuscin in the body tissues. Whether the changes in the liver and pancreas are due to the deposition of the pigment or to the same toxic agent which is supposed to cause the blood destruction is still a disputed question. It has been suggested that the excessive amount of iron in the tissues may be due to defective elimination rather than to increased formation from the blood.

ACUTE PULMONARY ŒDEMA, WITH SPECIAL REFERENCE TO A RECURRENT FORM.[1]

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The œdema of the lungs to which I desire to call attention is not that so commonly seen in the period of lost compensation of valvular or muscular heart disease, in cachectic states, in grave infectious diseases, or in acute and chronic nephritis in the terminal phase. It bears no relation to subcutaneous dropsy, and in typical cases is not associated with it. In the suddenness of its onset, in the alarming character of its symptoms, and in the mortal peril in which it places the patient, acute pulmonary œdema resembles angina pectoris, and deserves, with equal propriety, a separate place in our text-books. Those who, interested in the subject, look through the text-books and encyclopedias of medicine will be astonished to find how little mention is made of acute œdema of the lungs or, for that matter, of pulmonary œdema in general.

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[1] Read in abstract at the meeting of the Association of American Physicians, Washington, D. C., May 15 and 16, 1906.

History. Laënnec,[2] in 1819, alluded in his chapter on œdema of the lungs to a suffocative orthopnœa that sometimes carries off children after an attack of measles, and considered it an idiopathic anasarca of the lungs.

Andral,[3] in his commentary to the fourth edition of Laënnec's *Traité de l'Auscultation médiate,* clearly describes acute pulmonary œdema, and compares it with œdema of the glottis; but neither in this commentary nor in his own *Cours de Pathologie interne*[4] does he speak of the causes. He recognizes three forms of œdema: (1) A fulminant form (forme suraigue), in which the patient is suddenly seized with an intense dyspnœa that in a short time causes death; (2) a subacute form, in which the dyspnœa, though sudden in onset, lasts somewhat longer before death ensues; and (3) chronic œdema, which does not concern us at the present time.

Legendre,[5] in 1846, speaks of an acute pulmonary œdema occurring during the course of scarlet fever.

Rokitansky,[6] like Andral, compares acute pulmonary œdema with œdema of the glottis; it is, he states, a frequent cause of suffocation in adults and newborn children, and is often combined with hyperemia and serous effusion within the cavity of the cranium.

Bernheim,[7] in his clinical lectures, refers to a fulminant pulmonary œdema during the course of acute articular rheumatism; and Lebreton,[8] among the pulmonary complications of rheumatism, mentions an acute œdema of the type under consideration.

Landouzy[9] observed two rheumatic patients with pulmonary œdema, one having four and the other five recurrent attacks; neither had albuminuria.

Jaccoud[10] reports a case of typhoid fever terminating fatally within fifteen minutes from acute pulmonary œdema, which he designates as "un veritable coup de sang pulmonaire." The autopsy revealed nothing to explain the tragic event.

Debove[11] speaks of repeated accesses of acute pulmonary œdema, and cites the case of a physician having aortic disease who died in half an hour in an attack, after having had several before. Debove thinks that aortic affections, and cardiac diseases in general, predispose to acute pulmonary œdema.

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[2] Treatise on Diseases of the Chest and on Mediate Auscultation, 1830, p. 179.

[3] Laënnec's Traité de l'Auscultation médiate, 4ième édition, par Andral, t. xi, p. 424.

[4] Cours de Pathologie interne; 4ième édition, 1842, p. 123.

[5] Œdeme aigue du tissue cellulaire de poumon à la suite de la scarlatine, quoted by Fouineau.

[6] Manual of Pathological Anatomy, Sydenham Society's translation, iv, p. 71.

[7] Leçons de Clinique médicale, 1877, p. 494, quoted by Fouineau, q. v.

[8] Thèse de Paris, 1884, quoted by Fouineau.

[9] Bulletin médical, 1897, p. 445.

[10] Clinique de la Pitié, i, 1883, 1884, p. 567.

[11] Bulletin médical, 1897, xi, p. 400.

P. de La Harpe,[12] in 1881, reported three cases that illustrate the condition of acute pulmonary œdema very well: Case I. A woman, aged forty-three years, healthy, exposed herself to inclement weather after a slight attack of influenza; œdema of the lungs developed and caused her death within a short time. Case II. A woman, aged sixty years, also healthy, after one or two nocturnal attacks, died of œdema in a few minutes. Case III. A man had from fifteen to twenty attacks, all terminating by repeated eructations of gas and the voiding of large amounts of almost colorless urine. Similar cases are reported by Lund.[13]

One of the most important communications is that of Dr. Lindlay Steven,[14] of Glasgow, who, under the name of acute suffocative pulmonary œdema, reports two cases, in both of which there were evidences of renal disease; and in one of them there was also an affection of the aortic valve. Steven's paper stimulated the report of a number of other cases, one of the most interesting being that of Lissaman,[15] whose patient, a woman, aged forty-five years, had within two and a half years seventy-two attacks, all characterized by sudden onset, pain in the chest, frightful dyspnœa, lividity of the face, profuse cold sweats, labored action of the heart with small and rapid pulse, and the ejection, without cough, of enormous quantities of thin, pink, seromucous fluid. All the attacks were nocturnal.

Four well-studied cases have been reported by Hewlett,[16] who adopts Steven's designation of acute suffocative pulmonary œdema: Case I. A woman, aged forty-nine years, had six attacks—two in the afternoon, the others at night. Albuminuria, myocarditis, arteriosclerosis, and hypertension. Case II. A woman, aged fifty-two years, had repeated attacks, after each of which albumin and casts were present in the urine. In the intervals only casts were found. Heart hypertrophied; blood-pressure high. Case III. A woman, aged sixty-nine years, had ten attacks, all but one at night. Urine contained a trace of albumin; no casts. Case IV. An alcoholic woman, aged fifty-two years, who died in an attack of œdema.

A fairly typical case of the recurrent variety is reported in this country by Crummer.[17]

Among the most important systematic contributions to the subject from the clinical and pathological standpoint should be mentioned those of Huchard,[18] Dieulafoy,[19] Renaut,[20] Fuoineau,[21] Bouveret,[22] and Coplin.[23]

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[12] Rev. méd. de la Suisse Rom., 1881, p. 322.

[13] Lund. Norsk. Mag. for Lägevid, quoted by Huchard and by Fouineau.

[14] Lancet, January 11, 1902.

[15] Ibid., February 8, 1902.

[16] Intercolonial Medical Journal, 1903, p. 611.

[17] Northwestern Lancet, 1902, p. 203.

Its pathogenesis has been studied by Welch,[24] whose researches are of fundamental importance; by Sahli,[25] Grossman,[26] Löwit,[27] and others.

Etiology. Acute pulmonary œdema is associated with a variety of affections that, in a clinical sense, may be considered in the light of causes. I would classify them as follows: (1) Arteriosclerosis; (2) Bright's disease; (3) heart disease—angina pectoris; myocarditis; valvular affections; (4) asthma; (5) acute infectious diseases—typhoid fever, measles, rheumatic fever, influenza, pneumonia, etc.; (6) pregnancy; (7) paracentesis of thorax and of abdomen; (8) angioneurotic œdema; and (9) obscure conditions of questionable causal relationship (hysteria, etc.).

1. *Arteriosclerosis.* A large number of the reported cases of acute pulmonary œdema showed arterial disease. Case II of my series belongs to this group. Huchard[28] contends that vascular disease, especially aortitis and periaortitis, is the only cause of acute pulmonary œdema. No doubt it is an important factor in many cases, but not the essential one in all. Clifford Allbutt[29] admits the importance of aortitis; but very properly lays stress on the coexistence of general arteriosclerosis. The œdema results, according to Huchard, from disturbance in the innervation of the pulmonary vessels brought about by extension of the aortitic or periaortitic process to the cardiopulmonary plexus.

2. *Bright's Disease.* The important relationship that Bright's disease bears to acute pulmonary œdema was first clearly pointed out in 1890 by Bouveret, and afterward emphasized by Dieulafoy, Brouardel,[30] and others. Case I of my series is an illustration.

Dieulafoy, like Andral and Rokitansky, compares œdema of the lungs to œdema of the glottis, which is known to be one of the occasional complications of Bright's disease. How the nephritis acts has been much discussed by French writers: whether directly by toxemia (uremic poisoning) or indirectly through disease of the heart and aorta. For myself, I believe that nephritis can produce acute pulmonary œdema without the intervention of arterial or cardiac disease, through the mediation of poisons, the true nature of which remains unknown.

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[18] Bulletin médicale, 1897, xi, p. 393.

[19] Bulletin médicale, 1897, xi, p. 400.

[20] Bull. de l'Acad. de Méd., 1897, xxxvii, p. 557.

[21] L'Oedème du Poumon, Thèse de Paris, 1898.

[22] Rev. de méd., March, 1890.

[23] Therapeutic Gazette, April, 1906.

[24] Arch. f. path. Anat. und Phys., 1878, lxxii, p. 375.

[25] Arch. f. exper. Path. und Pharm., 1885, xix, p. 435.

[26] Zeit. f. klin. Med., xii, xv, xxi, 1887–1890.

[27] Ziegler's Beiträge, xiv, p. 401; Centralblatt f. allgem. Path. und pathol. Anat., 1895.

[28] Loc. cit.

[29] Lancet, July 8, 1903, p. 141.

[30] Bull. de l'Acad. de Méd., 1897, xxxvii, p. 509; Bulletin médicale, 1897, xi, p. 400.

3. *Heart Disease.* I have separated this from arteriosclerosis, with which it is generally associated, because there are cases of œdema in the literature in which the last condition is not mentioned, and may be presumed not to have existed, but in which there was valvular disease of the heart. It must be remembered that the œdema here considered is to be distinguished, as I stated in the beginning of the paper, from the ordinary œdema of valvular disease, which is a part of a general œdematous process.

With regard to angina pectoris, the coincident occurrence of œdema is noted but by few authors. It is presented in Case V of my series. Angina pectoris is, no doubt, often associated with aortitis, but not necessarily so; and certainly not always with evident peripheral arterial disease. In my own case there was no general arteriosclerosis. I have for that reason separated the pulmonary œdema of angina pectoris from that of arteriosclerosis.

4. *Asthma.* The occurrence of acute suffocative œdema during the course of asthma is illustrated by two of my cases—III and IV. Differentiation of the condition from a violent asthmatic paroxysm can be made by attention to the physical signs, the character of the expectoration, and the profound collapse. Moreover, the patients themselves are aware that something very different from an asthmatic attack is taking place. It is quite probable that many of the so-called asthmatic attacks coming on in middle or advanced life, without any previous history of asthma, are in reality attacks of pulmonary œdema.

5. *Acute Infectious Diseases.* In the section on history I have already cited a number of authors who have observed acute pulmonary œdema during (*a*) typhoid fever (Jaccoud and Merklen[31]); (*b*) rheumatic fever (Lebreton and Landouzy); (*c*) scarlet fever (Legendre); (*d*) influenza (Glasgow[32] and Lemoine[33]); (*e*) pneumonia (Souin de la Savinière[34]).

6. *Pregnancy.* The first to report a case of acute pulmonary œdema during pregnancy was Charles Wilson[35] (1856), whose patient was a woman, aged thirty-four years, eight months pregnant. There was anasarca and scanty, high-colored urine (no analysis); sudden pulmonary œdema at night; recovery after abstraction of twenty ounces of blood. Premature, spontaneous labor, followed by puerperal mania, with recovery under opium.

Vinay,[36] in 1896, reported three cases of acute pulmonary œdema during pregnancy, two of which were speedily fatal; in all three there was mitral-valve disease, and in one of them also nephritis. One of my cases, VI, belongs to this group.

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[31] La Méd. moderne, 1903, xiv, p. 145.

[32] Amer. Jour. Med. Sci., April, 1895, p. 425.

[33] Press méd., 1905, p. 151.

[34] Contribution à l'etude de l'œdème aigue du poumon, Thèse de Paris, 1873, quoted by Fouineau.

[35] Edinburgh Med. Jour., 1856, ii, part i, p. 481.

[36] Lyon méd., 1896, lxxxii, p. 289.

7. *Paracentesis of the Thorax and of the Abdomen.* In rare instances tapping of the chest, and more rarely still of the abdomen, is followed by the signs of acute pulmonary œdema, with the expectoration of large amounts of frothy albuminous material (albuminous expectoration). Originally it was believed that the albuminous expectoration was due to a wound of the lung, with a sucking up and the expectoration of the pleural fluid. This is not the case. The œdema results from an afflux of blood to the expanding lung, and a leakage of serum through the walls of the vessels, which by reason of long compression have become changed and rendered more permeable. In a paper upon this subject,[37] in which a bibliography will be found, I speak of this afflux as "congestion by recoil."

8. *Angioneurotic Œdema.* A case of sudden pulmonary œdema with coincident fugitive swelling of the face is reported by Hermann Müller,[38] who looks upon the pulmonary œdema as of the same origin, namely, angioneurotic, as of that of the face.

9. *Obscure Conditions of Questionable Causal Relationship.* Lavallée[39] reports a case of tabes dorsalis with two attacks of localized pulmonary œdema. There was arteriosclerosis, but no albuminuria and no valvular disease. In a case reported by Léopold-Lévy,[40] there was no other morbid condition than hysteria. Poppert[41] observed acute œdema of the lungs after ether anesthesia and collected seven similar cases from the literature.

PATHOGENESIS. Pulmonary œdema, whether acute or chronic, is brought about by leakage of serum through the capillary walls into the air vesicles and intervesicular tissues. In order that such leakage may take place, the pressure within the capillaries must be raised, or the permeability of the walls must be increased, or both factors must co-exist.

Many are the theories that have been advanced to explain the acute form of œdema. For purposes of clearness they may be classified as follows: (1) The periaortitic theory; (2) the cardiac theory; (3) the toxic vasomotor theory; (4) the infectious theory; and (5) the increased permeability theory.

1. *The Periaortitic Theory.* Huchard believes that the principal cause of acute pulmonary œdema is aortitis or periaortitis with extension of the inflammatory process to the cardiopulmonary plexus. The pathogenetic sequence, to quote his words, is as follows: "Disturbances of the innervation of the cardiopulmonary plexus by periaortitis; considerable augmentation of the vascular tension in the small circulation; acute or rapid insufficiency of the right ventricle." This is a satisfactory theory for those cases in which angina pectoris, Hodgson's disease, or other affections of the

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[37] AMER. JOUR. MED. SCI., April, 1902.

[38] Corresp. f. Schweizer Aerzte, 1891, p. 432.

[39] Journ. des Praticiens, May 14, 1897.

[40] Arch. de méd., 1895, clxxvi, p. 473.

[41] Deut. med. Woch., 1894, p. 719.

aorta exist; but it does not give an adequate explanation of those cases in which evidence of aortitis and general arterial disease is absent.

2. *The Cardiac Theory.* The cardiac origin of pulmonary œdema has its ablest defender in Welch, whose classical essay from the laboratory of Cohnheim has been misunderstood by more than one later investigator. As restated by Welch[42] in a recent communication, the theory is as follows: "Pulmonary œdema is due to a disproportion between the working power of the left ventricle of such character that, the resistance remaining the same, the left heart is unable to expel in a unit of time the same quantity of blood as the right heart. Such disproportion may be brought about by paralysis or by spasm of the left ventricle." The salient feature of this theory then is a difference in the amount of blood thrown out by the two ventricles; the left expelling a little less in a given time than the right. As a result some blood remains behind in the left auricle; this reacts upon the pulmonary veins, so that they cannot empty themselves completely; consequently, there is a rise of pressure in the pulmonary circulation to a degree sufficient to bring about a transudation of fluid through the capillary walls.

The attacks upon this theory by Grossmann, who has produced pulmonary œdema with muscarin, by Sahli, and by Löwit are answered by Welch in the communication referred to, and need not be taken up here. Meltzer[43] has ingeniously applied Welch's theory to an explanation of the pulmonary œdema following adrenalin injection. He holds that in consequence of the great obstacle caused by the considerable constriction of all the smaller blood-vessels of the body, the left ventricle is incapable of throwing out all the blood that it receives from the pulmonary veins, while, on the other hand, the right ventricle, stimulated by adrenalin, unloads with increased energy upon the lungs all the blood which the contracting vessels drive into it.

3. *The Toxic Vasomotor Theory.* According to this theory, which is advanced by Bouveret,[44] acute pulmonary œdema is due to the action of toxic substances on the pulmonary vasomotor mechanism. There is doubt among physiologists whether the pulmonary arteries possess a vasomotor nerve supply, although Francois-Franck[45] seems to have shown that such an one does exist, and Hofmann[46] considers it practically demonstrated that the pulmonary vessels have vasomotor nerves, though their influence is not great.

The toxic theory is useful in explaining the acute pulmonary œdema of some cases of Bright's disease, that of angioneurotic œdema, and, perhaps, that of pregnancy when there is no valvular or vascular disease.

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[42] Meltzer, Amer. Med., 1904, viii, p. 19, 59, 151, 191.

[43] Loc. cit.

[44] Loc. cit.

[45] Arch. de Physiol., 1895, p. 816, quoted by Hofmann in Handbuch der Physiol. des Menschen, Nagel, p. 298.

[46] Handbuch der Physiol. des Menschen, Nagel, p. 300.

4. *The Infectious Theory.* Caussade, Milhit, and Israel de Jong[47] have recently described a subacute œdema accompanying diverse pulmonary affections and possessing a more or less epidemic character. The expectoration is albuminous, frothy, not sanguinolent, and is easily differentiated from that peculiar to the disease, *e. g.*, bronchitis or pneumonia, with which the œdema happens to be associated. In the sputum the pneumococcus, usually of a virulent type, is present; whence the authors conclude that it is the cause of the œdema. Chloride retention and the neuroarthritic diathesis are predisposing factors.

The mere presence of the pneumococcus hardly possesses the importance the authors give to it, for such presence is to be expected when the œdema is an accompaniment of inflammations of the lungs that are known to be associated with this organism. The possibility of an infectious pulmonary œdema is, however, undeniable. It is probably present at some stage of all acute inflammations of the lung; but the type of œdema under consideration here does not bear the stamp of an infectious origin. The sudden onset of the attacks—their brevity, their subfebrile or afebrile character—militate against such a view.

5. *The Increased Permeability Theory.* Increased permeability as the sole cause of acute pulmonary œdema, as advocated, among others, by Martin,[48] is not adequate; but in conjunction with increased blood pressure it becomes an important factor, especially in the form of pulmonary œdema following paracentesis of the thorax.

PATHOLOGY. Renaut[49]—who is of opinion that both anatomically and clinically acute pulmonary œdema possesses an individuality that indicates a special pathogenesis—finds that the principal lesion is a serous inundation of the pulmonary alveoli. The alveolar fluid may be slightly fibrinous. White cells are abundant, sometimes to such an extent as to resemble a pneumonic exudate. That the entrance of the liquid into the alveoli has occurred violently under pressure is shown by the many points of rupture in the alveolar walls. The epithelium disappears within the limits of the œdema, having been forced off by the sudden eruption of fluid. The capillaries of the alveolar walls are flattened and rendered impermeable by compression, and, hence, are devoid of red cells. The large pulmonary and bronchial veins are, however, engorged with blood. The lymphatic spaces under the pleura are also distended.

Coplin,[50] whose study is the most recent, finds a finely granular deposit in the distended air vesicles and interstitial tissue, representing an albuminous fluid coagulated by the fixative. The capillaries usually show no conspicuous overdistention, and are frequently empty. The septal walls are often loosened and their tissues dissociated and infiltrated with mononuclear cells. The cell content of the air vesicles and interstitial tissue is subject to considerable variation; in some cases the erythrocytes are plentiful, and in others, not; polymorphonuclear leukocytes are at times abundant, and then bacteria are also present. In other cases again the cells are of the mononuclear type. Unlike Renaut, Coplin holds that detached vesicular epithelium can always be demonstrated. In some cases there is a marked swelling of the submucosa of the smaller bronchi and bronchioles, with a consequent narrowing of the lumen. The elastica is often stretched and the subserous and interstitial lymph vessels are distended.

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[47] Bull. et mém. de la Soc. méd. des Hôp. de Paris, February, 1906, p. 160.

[48] See Hewlett, Intercolonial Medical Journal, 1903, p. 611.

[49] Loc. cit.

[50] Loc. cit

Symptomatology. One of the most striking features of acute pulmonary œdema is the suddenness of its onset, often in what appears to be a state of good health. The patient is seized, usually at night, with a sensation of oppression and suffocation accompanied by a fear of dissolution. The breathing is labored and of a rattling character; the dyspnœa, or orthopnea, intense; the countenance either livid or pale and bedewed with sweat; cough may be harassing, or it may be entirely absent. In typical cases there is a copious, frothy, albuminous expectoration which may be expelled in gushes from the mouth and nose, and may reach the amount of one or two liters during an attack. Often it is blood-tinged, and in one of my cases (II) it had the appearance of raspberry vinegar; sometimes it is whitish like bronchial secretion. The action of the heart and the pulse are feeble in some cases, as in the two with asthma here reported; the pulse, however, may be full and bounding. Over the lungs the characteristic subcrepitant and bubbling rales of œdema are heard; not rarely, they are most abundant over the upper parts, while in the ordinary œdema they are most numerous over the lower lobes posteriorly.

The first attack of œdema may be fatal, like the first attack of angina pectoris. More frequently the first attack is not fatal, and is followed by numerous others, all presenting a striking uniformity and each placing the patient in imminent danger of death. For some reason the paroxysms are generally nocturnal, and not rarely they set in during sleep. The average duration varies from three to six hours; some cases last twelve to twenty-four hours; in the extremely fulminant types, as in Case I of my series, death may take place within ten or fifteen minutes, the patient literally drowning in his own juices.

Brouardel[51] has pointed out the medicolegal importance of these hyperacute cases in which sometimes there is no expectoration, death taking place by suffocation before the patient has a chance to rid himself of any fluid. As to the number of attacks, the case of Lissaman[52] with seventy-two seems to hold the record. It is important to bear in mind this tendency to recurrence, so as to give a reasonable prognosis, and to be prepared to treat the attacks on a definite, well-considered plan.

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[51] Loc. cit.

Cerebral symptoms may be present, as in Case II, in which aphasia and hemiplegia complicated the attack and outlasted it for some time. Delirium, convulsions, and coma have also been noted.

DIAGNOSIS. Acute pulmonary œdema must be distinguished from (*a*) asthma; (*b*) pulmonary embolism; (*c*) acute dilatation of the heart; and (*d*) uremic asthma.

Asthma. From asthma, acute pulmonary œdema may be distinguished by the fact that in the former there is very little expectoration during the attack, and is established only when the seizure has passed off, and is limited to a small amount of glairy material or a few mucous pellets. The piping rales of asthma are different from the mucous, bubbling rales of œdema.

Pulmonary Embolism. In pulmonary embolism there is an embolic source; expectoration is developed later than in acute pulmonary œdema, in which it usually coincides with the onset of dyspnœa; when once established in embolism it is less abundant and more definitely bloody than in pulmonary œdema.

Acute Dilatation of the Heart. The dyspnœa and cyanosis in acute cardiac dilatation are rarely as intense as in acute pulmonary œdema. There may be œdema of the lungs; the difference here is perhaps only one of degree. In typical cases of acute pulmonary œdema the dominant feature is the inundation of the lung with fluid, and the dyspnœa is more directly mechanical than in dilatation of the heart.

Uremic Asthma. The dyspnœa in uremic asthma is seldom as severe as in acute pulmonary œdema, nor is it accompanied by profuse frothy expectoration, and, hence, not by the characteristic physical signs of œdema of the lungs.

TREATMENT. Treatment must be prompt to be effective. The best results are obtained by venesection. Merklen,[53] with great enthusiasm, says, "c'est l'unique salut." The abstraction of a small amount of blood usually suffices, though in rare instances as much as from 400 to 600 c.c. has to be taken. When bleeding cannot be done, or in addition thereto, dry cups may be used. Half a dozen wineglasses, in which air has been rarefied by burning alcohol, should be placed over the back; and if the patient is turned on his side several can be put on the front of the chest at the same time. If there is much nervous excitement, and a terrifying feeling of impending death, a small dose of morphine should be administered hypodermically. Authors, it must be admitted, differ as to the value of morphine. Hewlett recommends it; Steven condemns it. Lissaman found chloroform eminently successful in his remarkable case. When the pulse is feeble and asystole is threatened, diffusible stimulants, such as whiskey, ammonia, and hypodermic injections of camphor, are useful. Crummer ascribes the favorable outcome in his case to adrenalin chloride; but as far as this drug is concerned, the experimental observations of Bouchard and Claude,[54] of Meltzer, and of Coplin, who found that it could produce œdema in the lower animals, ought to make us cautious. Nitroglycerin suggests itself as a remedy in those cases in which there is every evidence of arterial hypertension, namely, in the arteriosclerotic and renal types. Oxygen is an agent of value when there is much cyanosis.

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[52] Loc. cit.

[53] Loc. cit.

As I have said before, the likelihood of recurrence should be borne in mind, and should be made clear to the relatives of the patient. Where possible an intelligent member should be instructed in the method of applying dry cups, and, perhaps, in the use of the hypodermic syringe. Such precautions commend themselves more especially because the attacks generally occur at night, when it may be difficult to obtain medical aid at once.

REPORT OF CASES: CASE I.—Mrs. L., aged forty-six years; numerous attacks of acute œdema coming on without apparent cause. First attack in 1902, two days after death of mother; lasted one and one-half hours. No albuminuria in the beginning, later small amount of albumin, urine pale, low specific gravity, casts from time to time. Attacks characterized by dyspnœa, rattling breathing, mucous rales, sense of fear and terror, high tension pulse. Patient neurotic and subject to laughing and crying spells. Death occurred, December, 1905, in an attack lasting ten minutes, during which frothy, bloody fluid gushed from mouth and nose. Autopsy: Chronic interstitial nephritis, hypertrophy of the heart; intense pulmonary œdema, fresh petechial hemorrhages in the pleura and pericardium.

CASE II.—Mr. W., aged seventy-two years; always healthy. Seized suddenly one night with intense œdema of the lungs; chest filled with moist rales; reddish, frothy fluid gushing from mouth; dyspnœa, collapse, unconsciousness, paralysis of right arm and leg, pulse irregular, reduplication of first sound at apex. Marked arteriosclerosis, urine (catheter) albuminous. Disappearance of pulmonary signs in two or three days, palsy lasting a little longer; eventual recovery. Another attack a month later, January 23, 1904, not so severe; recovery. Numerous others, usually with stupor or coma and variable degrees of fleeting palsy and aphasia. Last attack, March 19, 1904, at 3 A.M.; intense dyspnœa, unconsciousness, fluid gushing from mouth and nose, paralysis of right arm; partial restoration of consciousness following venesection. Death, no autopsy.

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[54] Comp. rend. de l'Acad. des Sciences, December, 1902, p. 928.

CASE III.—Mrs. McC., aged forty years; asthma of several years' standing. During the past winter and spring repeated attacks of intense dyspnœa with rattling breathing, frothy expectoration, bubbling rales, alarming collapse, with pallor, feeble pulse, clammy skin. Attacks entirely different from asthmatic paroxysms, but occurring during asthmatic periods. A number of attacks within two or three months. None since going south.

CASE IV.—Miss I. C., aged twenty-five years; asthmatic. Without apparent cause seized at midnight with intense dyspnœa and fear of impending death; lips cyanotic, face ashen pale, skin clammy, no pulse at wrist, respiration rattling, chest filled with bubbling rales, frothy fluid slightly tinged with blood gushing from mouth and nose. Urine normal. Attack subsided by morning; extreme prostration for several days; one slight recurrence while still in bed.

CASE V.—Mrs. N. S., widow, aged fifty-eight years. For three or four years attacks of anginoid pain in precordium with radiation into left arm. Attacks always associated with orthopnœa and signs of pulmonary œdema; the latter at times confined to left lung, generally bilateral. Best results during attacks from dry cupping and nitroglycerin. Death from cardiac dropsy, no autopsy.

CASE VI.—Mrs. S., aged thirty-eight years; pregnant seven months —eighth pregnancy. Seized without warning and without previous exposure with an attack of intense dyspnœa and a feeling of oppression and anxiety. There was a great deal of wheezing and cyanosis, harassing cough, and alarming prostration. My first impression was that the patient had asthma; but antiasthmatic treatment had no result. The rales, which had been dry, soon became moist and bubbling, and a free, whitish, frothy expectoration developed. The patient had never had asthma nor any other illness; had passed through five labors and three miscarriages without a doctor, a midwife only attending. There was no history of previous kidney disease; nevertheless, the urine was loaded with albumin, but free from casts. The heart presented nothing abnormal; there was no arteriosclerosis. Her condition becoming worse, and as she seemed in danger of suffocation, Dr. E. E. Montgomery was called in consultation, and it was agreed between us that labor should be promptly induced. Under ethyl-chloride anesthesia an attempt was made to stretch the mouth of the womb, but it could not be done sufficiently, and it was thought best to introduce a piece of gauze and await developments. Free bleeding occurred during the stretching. To our surprise labor did not follow, but the patient rapidly improved, and within twenty-four hours had practically recovered from her dangerous condition. The urine cleared up entirely within a few days. Six weeks later, without any warning, the œdema returned. I promptly bled her from the elbow, with the most happy result. Her confinement is expected at any day.[55]

Conclusions. 1. There is an acute pulmonary œdema that comes on without any apparent exciting cause and often kills in a short time.

2. Recovery from an attack is frequent; but there is a striking tendency to recurrence, subsequent attacks being similar to the first. They set in suddenly, usually at night, and place the patient's life in jeopardy.

3. The clinical causes are various: arteriosclerosis and cardiac and renal disease are most important.

4. The pathogenesis is obscure; vasomotor disturbances and a disproportionate activity of the two ventricles (the right predominating) are the chief factors.

5. The chief symptoms are agonizing dyspnœa, cyanosis, cough, expectoration of frothy albuminous fluid, and profound prostration. Over the lungs, often more over the upper than the lower portions, the characteristic moist rales of edema are heard.

6. The most valuable remedies are bleeding, dry cupping, and cardiac stimulants.

7. The condition is of sufficient importance to warrant its having a special chapter in the text-books of medicine.

THE NATURE OF APLASTIC ANEMIA AND ITS RELATION TO OTHER ANEMIAS.[1]

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(From the William Pepper Laboratory of Clinical Medicine, Phoebe A. Hearst Foundation.)

Aplastic anemia was first described by Ehrlich[2] in 1888. Since that time isolated cases have been reported and recently the condition has received considerable attention. The following typical case occurred in the University Hospital in the service of Dr. Stengel, to whom I am indebted for the clinical data.

J. C., female, aged thirty-three years, white, married, was admitted to the University Hospital December 18, 1905, complaining chiefly of palpitation of the heart and vomiting after taking food.

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[55] Since writing this she was delivered without any difficulty by Dr. V. A. Loeb, to whom I transferred her for this purpose; she has had no other attacks.

[1] Read at the meeting of the Association of American Physicians, Washington, D. C., May 15 and 16, 1906.

[2] Charité Annalen, 1888, xiii.

Previous Medical History. The patient had measles, whooping-cough, and chicken-pox during childhood; diphtheria at twenty-two years; "congestion of the lungs" at twenty-five years. Menstruation began at eleven years, and has always been regular and at times painful. For the past eleven years, since her last child was born, the discharge has lasted about a week and has been profuse. The patient says that until the onset of the present illness she has always been robust.

Social History. The patient was married at the age of nineteen years, and is the mother of two healthy children, respectively twelve and eleven years of age. She had one miscarriage three years ago, apparently induced by severe exertion. Her work has been confined to household duties.

Family History. The patient's father died of "liver abscess" at the age of thirty-eight years. Her mother is living and well at fifty-seven years. Four sisters are living and well. There is in the family no history of tuberculosis, cardiac or renal disease, or neoplasm, and no condition similar to that of the patient.

History of Present Illness. The present trouble dates from about eight weeks ago, when the patient noticed that she was becoming pale, objects appeared yellow to her, and she was constantly fatigued. Six weeks ago she began vomiting after taking food. This vomiting was inhibited by her lying down. Shortly after the vomiting commenced cardiac palpitation and dyspnœa on the slightest exertion made their appearance, and the patient noticed that she was becoming progressively paler and weaker. She judges that in the past eight weeks she has lost about twenty pounds. During the last week she has had six attacks of temporary unconsciousness lasting about five minutes each. They were induced by no apparent cause. She has not fallen in the attacks, has not bitten her tongue, and has had no convulsive movements during the attack. Her appetite is poor and the bowels constipated. She has sour eructations and vomits on the slightest exertion or excitement. She has noted no discharge of blood from the mucous membranes. The feet have not been swollen.

Physical Examination. The patient is a large-framed woman with subcutaneous fat well preserved. The contrast between the degree of nutrition and the degree of anemia is very striking. The skin is of a lemon-yellow color, the mucous membranes are universally pale.

There are no areas of hemorrhage in the skin or visible mucous membranes. There is no enlargement of the thyroid, or of the cervical, axillary, or epitrochlear glands. No jaundice. Pupils are equal and react sluggishly to light. The patient complains of some loss of vision in the left eye. Teeth are imperfect and carious. Thorax is well-formed. Pulmonary resonance extends anteriorly from one inch above the clavicle to the lower border of the sixth rib in the midclavicular line on the right; to the middle of the fourth rib on the left; posteriorly on the right, to the tenth dorsal vertebra; on the left, to the angle of the scapula. Below the angle of the scapula the resonance is somewhat impaired down to the tenth dorsal vertebra. In this area fremitus is diminished and breath sounds are feeble. No rales are heard. This area of diminished breath sounds extends anteriorly to the midaxillary line. In the remaining pulmonary area the breath sounds and fremitus are normal. The usual band of resonance over the shoulder is present on both sides. The physiological difference in breath sounds between right and left apex is less marked than usual.

The right border of the heart dulness is at the right border of the sternum; upper border, middle of fourth rib; left border, one inch outside of the left midclavicular line in the fifth interspace. The apex-beat is in the fifth interspace three-fourths of an inch inside of the midclavicular line. No thrills are present. There is a soft systolic murmur at the apex, heard also at the base, and most intense at the fourth interspace one inch to the right of the sternum. There is a slight accentuation of the pulmonic second sound. There is a continuous murmur heard over the innominate artery resembling a venous hum in its rhythm. There is also a dull venous hum high in the neck, over the jugular vein.

The abdominal walls are thick, but the abdomen is neither distended nor retracted. The liver dulness extends from the lower border of the sixth rib to within one inch of the costal margin in the midclavicular line. It is not palpable. Neither kidney is palpable. No tumor is discovered in the abdomen. The spleen is not enlarged, extends from the eighth to the tenth rib. It is not palpable. Nervous examination reveals no irregularities of reaction or sensation.

Urine Analysis. Amber, clear, slightly acid; specific gravity, 1010; slight trace of albumin; no sugar.

Gastric Analysis. Free HCl, 36. Total acidity, 64. No lactic acid. No Oppler-Boas bacilli.

Feces Analysis. Stool is yellow, hard, and alkaline. Contains considerable vegetable detritus, a small amount of casein, triple phosphates, and a small amount of soap. Bile is present. There is no occult blood and no indication of the presence of parasites.

Blood Examination. Hemoglobin, 10 per cent.; red blood corpuscles, 800,000; white blood corpuscles, 2560; color index, 0.62. Differential count: lymphocytes, 67 per cent.; polymorphonuclear neutrophiles, 27 per cent.; polymorphonuclear eosinophiles, 0.4 per cent.; large mononuclear leukocytes, 3.5 per cent. No myelocytes were found. In counting over 1000 leukocytes, one normoblast was found. Despite repeated searches no megaloblasts were found. There were no microcytes. Very slight poikolocytosis, and no anisocytosis were found. Subsequent examination of the blood revealed but trivial variations from this count.

Ophthalmoscopic examination revealed a well-marked, low-grade neuroretinitis, with numerous hemorrhages of various size and shape lying in the nerve-fiber layer, and a few more deeply placed hemorrhages. The swelling of the left nerve head and obscuration of its edges was more marked than in the right eye.

Without the appearance of any new noteworthy symptoms, the patient grew progressively weaker and died with symptoms of œdema of the lungs eight days after admission, or approximately nine weeks after the onset. Permission for the autopsy, performed four days after death, was obtained through the efforts of Dr. F. H. MacFarland, to whom I am deeply indebted.

Autopsy. The macroscopic findings briefly stated were as follows: The body of a well-formed, well-nourished, adult female. Height approximately five feet three inches. Weight about one-hundred and thirty-five pounds. Subcutaneous fat moderately abundant, light yellow in color. Muscles of fair consistency and pale. The left pleural cavity contained 400 c.c. of serosanguineous fluid; the right pleural cavity approximately 200 c.c. of fluid of the same character. There was œdema of the lungs and fatty degeneration of the heart muscle. There were no valvular defects. In the pelvic peritoneal cavity were 200 to 300 c.c. of dark-red, fluid blood containing one small clot. The liver was anemic and fatty; the spleen pale, firm, and not enlarged; the kidneys pale and fatty. Aside from pallor of the mucous membranes the gastrointestinal tract presented no abnormalities. There were no evidences of the presence of parasites. In the mesentery and retroperitoneal tissues a few hemolymph nodes were found, light reddish-brown in color, and of the size of small beans. The marrow of the ribs was light yellow in color; that of both the diaphysis and epiphysis of the tibia, fatty and extremely pale.

Histological Examination. There was fatty degeneration of the heart muscle in the form of extremely fine globules of fat, and a considerable hemosiderin deposit, mostly in the protoplasm of the cells at both poles of the nucleus. The section of liver showed a marked fatty change in the form of large globules confined almost exclusively to the central half of the individual lobules. There was an extensive deposit of hemosiderin occurring diffusely throughout the liver cells. In the section of spleen, there was noted a moderate proliferation of endothelium within the pulp. The follicles were prominent but apparently not hypertrophied. A few normoblasts were visible in the section. In the kidney the epithelium of the tubules of the cortex showed some disintegration and in many areas was detached from the basement membrane. Numerous small fat globules were present in the epithelium of the tubules, especially toward the free margins of the cells. The lumina were filled with a granular exudate including, here and there, shadows of red blood corpuscles. The hemolymph glands showed the peripheral sinuses to be small and there was some endothelial proliferation in the pulp. Smears and sections were made of the bone-marrow of the shaft of the tibia near the epiphysis. They both showed a large amount of fat, a very few normoblasts and normocytes, an absence of megaloblasts, a moderate number of lymphocytes, and a few myelocytes. No polymorphonuclear leukocytes could be identified.

I have succeeded in collecting from the literature ten cases in which the clinical phenomena and postmortem findings warrant their being considered cases of aplastic anemia. They are as follows:

Case I.—Bloch.[3] Female, aged sixty-three years; first symptoms noted, weakness and pallor; duration, six weeks. Blood: hemoglobin, 28 per cent.; red blood corpuscles, 1,970,000; leukocytes, 3250. Differential count: lymphocytes greatly preponderating; no nucleated red blood corpuscles; a few microcytes; no megalocytes; color index, 0.71. Postmortem findings: marrow of long bones showed no lymphoid hyperplasia; in marrow of ribs there were no megaloblasts. Prominent clinical feature: four days before death hemorrhage from buccal mucous membrane.

Case II.—Bloch.[4] Female, aged fifty-three years; first symptom noted was weakness. Blood: severe anemia, marked reduction in leukocytes, those persisting being almost entirely lymphocytes. No megaloblasts; after prolonged search a few normoblasts were found. Postmortem findings: marrow of shafts of long bones pale and fatty. Marrow of epiphyses of normal red color. Prominent clinical feature: numerous gastric hemorrhages occurred during the course of the disease.

Case III.—Ehrlich.[5] Female, aged twenty-one years; metrorrhagia eighteen days before onset. Blood: hemoglobin, very low; red blood corpuscles, 213,360; white blood corpuscles, greatly reduced. Differential count: lymphocytes, 80 per cent.; polymorphonuclear neutrophiles, 14 per cent.; large mononuclear leukocytes, 3 per cent.; eosinophiles, 0; no nucleated red blood corpuscles. Postmortem findings: bone-marrow yellow in shafts of long bones, slightly pinkish in diaphyses. No megaloblasts. No normocytes. Very few normoblasts and myelocytes. No polynucleated granulated cells.

Case IV.—Engel.[6] Female, aged thirty-one years; shortly before onset blood noted in stools and hemorrhage from gums; duration, three weeks. Blood: hemoglobin, 18 per cent.; red blood corpuscles, 2,115,000; white blood corpuscles, greatly reduced. Differential count: small lymphocytes, 90 per cent.; polymorphonuclear neutrophiles, 7 per cent.; no microcytes; no macrocytes; no nucleated red blood corpuscles. Color index, 0.42. Postmortem findings: diaphyses and epiphyses of long bones yellow and fatty. Microscopically, nothing but fat. Marrow of ribs gray and watery. No cells present. Prominent clinical feature: there was a doubtful history of swallowing of corrosive shortly before the onset of symptoms. Chief complaint was weakness.

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[3] Ziegler's Beiträge, 1903, xxiv, 231.

[4] Ibid.

[5] Loc. cit.

[6] Ztschr. f. klin. Med., 1900, ii.

Case V.—Evans and Halton.[7] Male, aged thirty-six years; first symptoms noted shortly after bleeding from nose lasting two and one-half hours; duration, two months. Blood: hemoglobin, 10 per cent.; red blood corpuscles, 770,000; white blood corpuscles, 2300. Differential count: polymorphonuclear neutrophiles, 6 per cent.; small lymphocytes, 90 per cent.; large lymphocytes, 4 per cent.; eosinophiles, none; no nucleated red blood corpuscles. No marked poikilocytosis. Shortly before death two nucleated red blood corpuscles were found; color index, 0.65; actual number of lymphocytes, 2070. Postmortem findings: skull bones showed fatty marrow with very few cells. Prominent clinical features: chief complaint was weakness and faintness.

Case VI.—Hirschfeld.[8] Female, aged twenty-eight years; onset shortly after delivery; duration, nine months. Blood: red blood corpuscles, 1,200,000; white blood corpuscles, 3600. Differential count: small lymphocytes, 72 per cent.; no eosinophiles; no nucleated red blood corpuscles; actual number of lymphocytes, 2592. Postmortem findings: bone-marrow universally yellow; bone-marrow was composed chiefly of small lymphocytes, a very few granulated cells, and a few normoblasts. Prominent clinical features: numerous small hemorrhages into the skin, mucous membranes, and retina occurred during the course; menstruation was extremely profuse.

Case VII.—Muir.[9] Male, aged fourteen years; the first symptoms noted were vomiting of blood, bleeding from nose, and hemorrhages into skin; duration, eleven weeks. Blood: hemoglobin, 12 per cent.; red blood corpuscles, 800,000; white blood corpuscles, 7000. Differential count: lymphocytes, 70 per cent.; polymorphonuclear neutrophiles, 25 per cent.; no nucleated red blood corpuscles; color index, 0.75; actual number of lymphocytes, 4900. Postmortem findings: bone-marrow fatty; very few nucleated red blood corpuscles or other normal cells of bone-marrow.

Case VIII.—Pasteur.[10] Male, aged twenty-four years; first symptom was pain in epigastrium; duration, seven weeks. Blood: hemoglobin, 20 per cent.; red blood corpuscles, 1,090,000; white blood corpuscles, 2600. Differential count: lymphocytes, 74 per cent.; polymorphonuclear neutrophiles, 20 per cent.; hyaline and transitional cells, 6 per cent.; no megaloblasts; color index, 0.91; actual number of lymphocytes, 1924. Postmortem findings: bone-marrow, pale; no polymorphonuclear leukocytes.

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[7] Jour. Amer. Med. Assoc. 1905, i, 1195.

[8] Deut. med. Woch., 1904, xxii, 650.

[9] Brit. Med. Jour. September 29, 1900.

[10] Lancet, November 21, 1903.

Case IX.—Zeri.[11] Female, aged fifty-seven years; first symptoms noted were hemorrhages into skin; duration, six months. Blood: hemoglobin, 20 per cent.; red blood corpuscles, 1,300,000; white blood corpuscles, 3580. Differential count: polymorphonuclear leukocytes were scarce in comparison to lymphocytes; no macrocytes; no megaloblasts; two normoblasts were found; color index, 0.76. Postmortem findings: marrow of long bones yellow in both diaphysis and epiphysis; the same in costal and vertebral bones. Prominent clinical features: during the course of the disease patient had prolonged epistaxis and numerous hemorrhages into skin.

Case X.—Zeri.[12] Male, aged forty-six years; onset preceded by diarrhœa; duration, three months. Blood: hemoglobin, 38 per cent.; red blood corpuscles, 1,423,000; white blood corpuscles, 2700. Differential count: polymorphonuclear leukocytes and transitional cells, 57 per cent.; eosinophiles, 0; later moderate poikilocytosis; a few microcytes and macrocytes; no nucleated red blood corpuscles; color index, 1.33; actual number of lymphocytes, 1161. Postmortem findings: marrow of flat bones, orange yellow; of long bones, in both epiphysis and diaphysis, yellow and fatty; scarcity of polymorphonuclear leukocytes; normoblasts with multiform nuclei were present; in the marrow of the shafts of the long bones degeneration and proliferation of connective tissue.

Making a composite case from these ten cases and my own, we obtain one of the following characteristics: age, thirty-seven years; duration, three months. Blood: hemoglobin, 18 per cent.; erythrocytes, 1,116,600; leukocytes, 3574; lymphocytes, 73 per cent.; actual number of lymphocytes, 2393; color index, 0.77. It is readily seen that the more prominent features of such a case are sufficiently marked to furnish a very striking clinical picture, associated with unique postmortem findings.

The disease, as characterized by the cases quoted shows itself to be first manifested by symptoms common to any profound anemia: ready exhaustion, dyspnœa, disturbance of vision, tinnitus, nausea, and vomiting. In a strikingly large percentage of cases, considerable or numerous small hemorrhages occur shortly before, or coincident with, the onset of the symptoms of anemia, as noted by the patient. The patient usually remains fairly well nourished as far as the bulk of the fixed tissues, particularly the fat, is concerned, and there is a striking disharmony between the quality of the patient's blood and state of bodily nourishment. Usually without any prominent symptoms referable to any other system or function,

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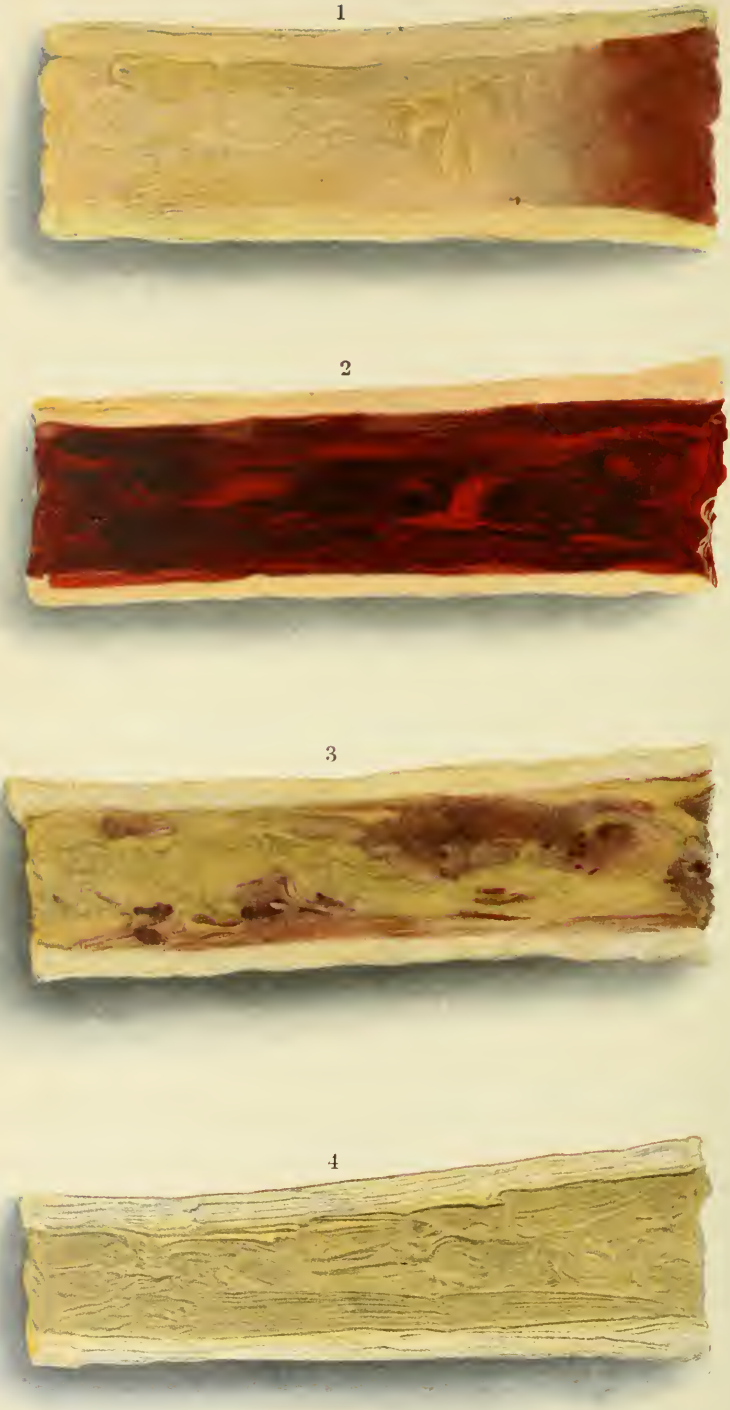
[11] Il Policlinico, s. m. fasc. vii, 1905.

[12] Ibid.



Bone Marrows.—1. Normal. 2. Typical progressive pernicious anemia. 3. Scattered areas of megaloblastic degeneration in progressive pernicious anemia. 4. Aplastic anemia.

the patient grows progressively weaker, and in from two to four months dies. Naturally the most noteworthy picture is afforded by the blood analysis, in which there is a profound reduction in the amount of hemoglobin and a somewhat less marked reduction in the number of red blood corpuscles, an absence or a remarkably small number of normoblasts, a total absence of megaloblasts, and a leukopenia associated with a relative lymphocytosis.

At autopsy, aside from the findings purely incidental to the grade of the anemia, there is the characteristic feature in the bone-marrow, which is intensely pale and fatty and lacks the evidences of blood regeneration.

A critical review of these more prominent symptoms immediately suggests the question that has been the source of some argument in regard to aplastic anemia, namely, its relation to progressive pernicious anemia. The onset of the disease, its course, excepting as to the rapidity of a lethal termination, the absence of any marked loss of substance in the fixed tissues, and the great reduction in the amount of hemoglobin and the number of red corpuscles, are common to both conditions. In our attempt to determine the relation between aplastic anemia and progressive pernicious anemia, let us discuss individually the phenomena at variance in the two conditions. Probably the most striking of these, and surely the most important, is the blood picture in which we find an almost complete absence in aplastic anemia of nucleated red blood corpuscles and the total absence of megaloblasts. Since the red corpuscles are a product of the bone-marrow, a consideration of them will necessarily involve a consideration of the bone-marrow. There is by no means a unity of opinion as to the primary effect of the etiological agent in pernicious anemia in its relation to the bone-marrow. Rindfleisch and Cohnheim[13] look upon the bone-marrow changes as primary, while others (Neumann, Litten, Orth) consider them as secondary, in the nature of an attempt on the part of the bone-marrow to compensate for the destruction of red blood corpuscles. Just as the character and number of the atypical red blood corpuscles vary in different cases of anemia, so does the character of the bone-marrow differ (see accompanying plate). In one case there is a red jelly-like marrow (megaloblastic degeneration) composed almost entirely of megaloblasts and associated with the presence of numerous megaloblasts in the circulating blood; in another case, but few megaloblasts and correspondingly little red marrow in the shafts of the long bones. In a case that I recently autopsied, there were a very moderate number of normoblasts and megaloblasts, and at the autopsy the shafts of the long bones were found to be fatty with islands of red scattered here and there through it. It is thus seen that deductions as to the condition of the bone-marrow can frequently be drawn from a consideration of the cells of the circulating blood.

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[13] Virchow's Archiv, lxvii, 291.

What of these relations in aplastic anemia? I have said that the blood shows an extremely limited number of nucleated red blood corpuscles, and we would consequently expect to find, as we in fact do, a pale bone-marrow in which the evidences of new blood formation are entirely wanting. We see, then, that in aplastic anemia there is a blood picture which is but a reflection of the condition of the bone-marrow, in which evidences of regeneration are entirely lacking, but in which there is no indication that the determining factor in the causation of the disease acts in any way different from that of the usual type of progressive pernicious anemia. It is but a difference in response of the organism to the causative agent.

What of the color index which in typical progressive pernicious anemia is considered to be characteristically high, and in aplastic anemia has been in all cases, excepting one, considerably below 1, and averages in all the cases 0.77? In progressive pernicious anemia, aside from megaloblasts, there are non-nucleated red blood corpuscles of considerably more than normal size, megalocytes. Laache has offered the usually accepted explanation for the high color index in progressive pernicious anemia in the large number of these abnormal cells present, which containing more hemoglobin than the normal cell naturally raise the color value. As has been remarked, in aplastic anemia there is usually little anisocytosis, and with the absence of both megaloblasts and megalocytes, it is readily seen why, on the basis of Laache's theory, the color index should not be raised above the normal. An interesting theory for the high color index in progressive pernicious anemia is proposed by Schaumann.[14] He considers that some of the iron deposited as hemosiderin in the liver and spleen is taken up by the existing corpuscles, used as a nucleus for the formation of more hemoglobin, and thus each individual corpuscle bears more than the normal amount. Some weight is lent to this view by experiments of Tallqvist.[15] In experimental anemias in dogs he showed that the color index was higher in cases of slow growing anemia than in those of rapid growth, and in the former the siderosis was greater than in the latter. This theory would also explain the low color index in cases of aplastic anemia, as they have almost all been of extremely rapid course.

Let us consider for a moment this more rapid course and invariably fatal termination of aplastic anemia with the course and termination in progressive pernicious anemia. We have seen that in aplasic anemia destructive forces are at work as in progressive pernicious anemia, but the regenerative process present in the latter is wanting in the former. If a destructive process be active and there be no constructive process to counteract it, it is but a natural consequence that, other things being equal, exhaustion will more readily result than if there be constructive changes.

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[14] Berl. klin. Woch. 1899, Nos. 1 and 3.

[15] Ueber experimentelle Blutgiftanæmien, Berlin, 1900.

Another feature at variance in the two conditions is the number of leukocytes and the proportions to one another of the various forms of leukocytes. In uncomplicated cases of progressive pernicious anemia the leukocytes average from 5000 to 6000; in aplastic anemia they average 3574. What is even more striking than this difference is the difference in the percentage of lymphocytes. In progressive pernicious anemia the lymphocytes vary from 37 per cent. (Strauss and Rohnstein[16]) to 31 per cent. (McCrae), while in aplastic anemia they average 73 per cent. A discussion of the principles underlying this variance must be prefaced by a few words concerning the origin of leukocytes. It is generally conceded that the granulated leukocytes, *i. e.*, the polymorphonuclear leukocytes and their progenitors, the myelocytes, have their origin in the bone-marrow; but there has been a vigorous discussion existing for some time as to whether the lymphocytes be the product of the bone-marrow or of the other more distinctly lymphatic structures, lymph glands, spleen, and lymphatic tissue of the gastrointestinal tract. Ehrlich believes firmly in the origin of the lymphocytes from true lymphatic structures, while others claim them to be of common origin with the granulated cells. That lymphocytes are formed in the bone-marrow there can be little doubt, for wherever lymphoid tissue is present, lymphocytes are produced; and, as was shown by Arnold and Ribbert, there is an element of lymphatic tissue in almost every organ of the body. So although lymphocytes may be produced in the bone-marrow by reason of the participation of the bone-marrow in the general distribution of lymphatic tissue, I do not believe that the production of lymphocytes is a specific function of it. Let us observe what bearing the lymphocytes in aplastic anemia have upon the question. We have seen that the red blood corpuscles and the granulated leukocytes, both admittedly products of the bone-marrow, are enormously reduced. But what of the lymphocytes? We have seen that in the average case of aplastic anemia there are 2393 lymphocytes, and it will be observed that this differs but slightly from the average normal number of lymphocytes, about 2200. Does it not seem irrational that if the lymphocytes be an integral part of the bone-marrow they should be so slightly affected and the other bone-marrow elements so profoundly reduced? As to the relation between the percentage of lymphocytes in progressive pernicious anemia as compared with that of aplastic anemia, I look upon it that in neither condition are they actually materially affected. In progressive pernicious anemia they are, on the average, approximately 35 per cent. of the total number of leukocytes, 5000 to 6000, which is but little less than 73 per cent. of 3574, the total average number of leukocytes in aplastic anemia. The difference depends entirely upon the variations in the number of polymorphonuclear neutrophiles and in harmony with the generally more intense character of the lesions in aplastic anemia they are more markedly reduced than in the former.

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[16] Die Blutzusammensetzung bei den verschiedenen Anämien.

In regard to the autopsy findings the essential differences in the two diseases lie in the bone-marrow, which, as we have attempted to show in the above analysis, manifests a complete failure of regeneration on its part.

Basing our deductions upon the above comparison, we thus see that progressive pernicious anemia is associated with regenerative processes which in aplastic anemia are lacking. As to the destructive processes, it has been shown by Hunter, Quincke, Pepper and Stengel that progressive pernicious anemia is primarily a hemolytic disease. That aplastic anemia is of the same nature, may be accepted by reason of the consistent occurrence of hemorrhages in practically all of the cases reported, and by reason of the hemosiderosis present in our case. It is unfortunate that in the previously reported cases no mention is made of this condition. We see, then, that the blood picture in aplastic anemia and the course of the disease are the result of two factors: the one the hemolysis, the other the failure of regenerative changes to compensate for this destruction. The failure of regeneration by the bone-marrow may be dependent upon one of the following three factors: first, a simple deficiency of the regenerative powers; second, an inhibitive action on the bone-marrow by the factors producing the destruction of the blood elements; third, a true aplasia of the bone-marrow. If the latter be the case, the aplasia is probably of recent origin, for if it were of long duration there would, in all probability, have been manifestations of a deficiency in blood formation before the advent of the hemolytic agent.

It will be necessary to discuss but briefly the relation that aplastic anemia bears to simple anemia. Lazarus looks upon aplastic anemia as a variety of simple anemia, basing his view upon the fact that aplastic anemia is most like simple anemia in the blood picture. A review of the cases that we have collected reveals the fact that in practically none of the cases has there been the history of the various organic diseases so frequently associated in an etiological way with simple anemia. It is true that hemorrhages do appear to have been more frequent and probably more profound in aplastic anemia than in progressive pernicious anemia, but that they stand in an etiological relation to aplastic anemia can scarcely be entertained. They have occurred throughout the course of the disease rather than precedent to it, and as Birch-Hirschfeld[17] has emphasized, the severity of the anemia stands in marked contrast to the amount of hemorrhage, though it must be admitted that in some cases when hemorrhages are small and numerous, and especially when they occur in the gastrointestinal tract, a calculation of the amount of blood lost is difficult or impossible; but the most important elements differentiating aplastic anemia from simple anemia are furnished by a consideration of the clinical features of aplastic anemia. It is an essentially pernicious disease in course, duration, and termination, and serves well to illustrate the futility of attempting the diagnosis of most blood diseases on the blood picture alone.

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[17] Verhandl. d. Congress f. innere Medizin, Wiesbaden, 1892, xi.

The exclusion from our list of a number of cases that have been reported as cases of aplastic anemia, or present otherwise features suggestive of this disease, requires some words of justification.

Senator[18] reports the case of a girl, aged thirteen years, complaining, chiefly of weakness and vertigo. The disease was of three months' duration, and the blood findings were as follows: hemoglobin, 8 to 10 per cent.; erythrocytes, 1,040,000; leukocytes, 8000; poikilocytosis; no nucleated red blood corpuscles; no eosinophiles. The majority of the white blood corpuscles were lymphocytes. Blood from the heart, taken at autopsy, showed 728,000 red blood corpuscles and 11,200 white blood corpuscles, of which 83.6 per cent. were lymphocytes. At autopsy the bone-marrow was found to be universally red, showing microscopically large numbers of lymphocytes and a few normoblasts. Two elements in this case tend to exclude it from the aplastic anemias. The one is the metaplastic bone-marrow, containing numerous lymphocytes, the other the occurrence clinically of a considerable actual increase of the lymphocytes, as is shown by the fact that 83.6 per cent. of 11,200 leukocytes were lymphocytes. Senator himself says of the case, after a consideration of the clinical and postmortem features, "we are thus dealing with a case of myelogenous pseudoleukemia, and according to the present-day terminology one of a pure medullary form, since the lymphatic glands were not at all, and the spleen to a very moderate degree, swollen."

Blumer[19] reports a case of twenty-two months' duration, occurring after the loss of considerable blood from hemorrhoids. The blood showed 20 per cent. of hemoglobin, 1,516,000 red blood corpuscles, and 4000 white blood corpuscles, of which 88.3 per cent. were lymphocytes. An average of one normoblast to a field was found. At autopsy the bone-marrow of the upper part of the tibia was yellow, that of the ends of the other long bones partly red and partly fatty. On the basis of a duration of twenty-two months, which is more than seven times the average duration, and the presence of a considerable number of normoblasts, in harmony with which is the partly red and partly yellow bone-marrow of the diaphyses, we feel justified in looking upon this as a doubtful case of aplastic anemia. It has more of the features, excepting for the percentage of lymphocytes, of a low grade normoblastic progressive pernicious anemia.

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[18] Ztschr. f. klin. Med., lix, 1.

[19] Bull. Johns Hopkins Hospital, April, 1905.

Schaumann's[20] case, which he reports as aplastic anemia, is very suggestive, but the data furnished in the report of the case are insufficient to warrant the diagnosis.

The case of Pane,[21] quoted by Zeri as a case of aplastic anemia, is doubtful. The meager clinical data furnished, associated with the finding postmortem of a splenic tumor, and hyperplasia of Peyer's patches and the solitary follicles of the intestinal tract, do not justify us in considering the case one of aplastic anemia.

Wolff's[22] case presents, as he himself says, "more features of an aplastic leukemia than an aplastic anemia."

Escherich[23] reports a case of anemia in a child, aged four years, that strongly suggests aplastic anemia. The disease was of six months' duration. The red blood corpuscles varied between 575,000 and 737,500; the hemoglobin between 10 and 15 per cent.; the leukocytes between 3500 and 7000. The statement is made that the majority of white blood corpuscles were lymphocytes. There was considerable poikilocytosis and macrocytosis and no nucleated red blood corpuscles. There was at first some swelling of the lymphatic glands in the region of the mastoid and in the left axilla. It is to be regretted that there was no postmortem examination to confirm the strong suggestion of its having been a case of aplastic anemia.

Kurpjeweit[24] has recently reported two cases of anemia in the aged, which present some features suggestive of aplastic anemia, especially the first case. However, the greatly enlarged spleen, measuring 18.5 cm. x 12.5 cm. x 6 cm., and the but moderately severe course of the disease, together with the lack of leukocytic examination in the case, will not permit of a diagnosis of aplastic anemia. The second case also had a remarkably large spleen, 800 grams. Lymphocytes did not preponderate, and the bone-marrow showed normoblastic regeneration.

Conclusions. 1. Aplastic anemia is a variety of progressive pernicious anemia.

2. The essential features of aplastic anemia are: a rapidly fatal course; a marked reduction in the number of red blood corpuscles; a greater proportionate reduction in the amount of hemoglobin, resulting in a low color index; a leukopenia with a relative lym-

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[20] Volkmann's Sammlung klinischer Vorträge, n. f. No. 287.

[21] Riforma Medica, 1900, xvi, No. 4.

[22] Berl. med. Woch., 1905, xlii, No. 2.

[23] Wien klin. Woch., 1892, Nos. 13 and 14.

[24] Deut. Archiv. f. klin. Med., lxxxii, Nos. 5 and 6.

phocytosis; an absence of megaloblasts, and usually normoblasts. Postmortem: the characteristic finding is a pale bone-marrow in which the signs of erythrocytic and granulated leukocytic formation are wanting.

3. The differences between aplastic anemia and the usual form of progressive pernicious anemia result entirely from the absence of regenerative processes in the former.

4. The blood picture in aplastic anemia is the result of two factors, one the hemocytolysis, and the other the failure of regeneration on the part of the bone-marrow.

5. The failure of regeneration of the blood elements of the bone-marrow represents the result of one of the three following conditions: (*a*) A simple deficiency of the regenerative powers; (*b*) an inhibitive action on the bone-marrow by the factors producing the destruction of the blood elements; and (*c*) a true aplasia of the bone-marrow. If there be a true aplasia it is probably of recent origin, for if it were of long duration there would in all probability have been manifestations of a deficiency in blood formation before the advent of the hemolytic agent.

6. The relations of lymphocytes to leukocytes and red blood corpuscles in aplastic anemia lend evidence to the view that lymphocytes are not a specific product of the bone-marrow.

STUDIES IN THE NATURAL AND ARTIFICIAL INHIBITION OF PEPTIC DIGESTION.

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No matter how important the secretion of hydrochloric acid may be for the purpose of preparing the food for the action of pepsin, and for stimulating the production of pepsin and the secretion of the pancreatic, and probably of the intestinal juices, there can be no question, we think, that the actual work in the stomach of preparing the proteids for assimilation depends upon the peptic activity. Therefore it is exceedingly important in all cases of gastric disease to determine whether the peptic activity is normal or not, and whether, if it is normal under favorable experimental conditions, there may not be present in the gastric contents inhibitory factors which prevent, or make imperfect, the peptonization of the food.

A number of methods have been suggested for estimating the peptic activity. Of these the only ones of value clinically are those of Hammerschlag and Mett. Both are good, and give results that are useful in diagnosis and for the purpose of controlling treatment. We have elsewhere stated our reasons for preferring the method of Mett. Our technique in the use of this method has already been described. It yields results that are practically the same as those of other investigators.

The object of the present investigation was to determine, first, the natural inhibitory factors in the gastric contents; second, the inhibitory action of certain substances, chiefly those used as food preservatives.

The natural factors may be substances secreted by the stomach itself, or products, either normal or abnormal, of the digestive activity. Danielewsky claims to have discovered a substance, antipepsin, which exists in the gastric mucosa. It inhibits the action of pepsin but does not destroy it; it may be extracted with weak acid solution; it is not destroyed by heating to 70° C. or even by brief exposure to 100° C. Hensel found that it is not precipitated by phosphotungstic acid. It is organic in nature, but not an albumin. A similar substance is obtained from various organs. Weinland and Schwartz have also studied this subject. The latter, by boiling a solution of pepsin, was able to demonstrate that it acquired inhibitory properties as a result of destruction of the pepsin and immunity of the antipepsin.

Nearly every type of food, the carbohydrates, the hydrocarbons, the proteids, and the products of their digestion, and the various mineral constituents, have been accused of inhibitory activity.

The method has undergone some trifling modifications in the course of the work. At first we used rather strong solutions of pepsin. In a quantity of 16 c.c., 5 c.c. consisted of 2 per cent. pepsin, and the remaining 11 c.c. of a dilution of the substance under examination, or, for the purpose of control, $\frac{n}{20}$ HCl. The pepsin was dissolved in $\frac{n}{20}$ HCl and the substance to be tested in the same solution, excepting in the case of liquid substances bulky enough to make it necessary to acidulate them to the same degree with concentrated HCl. The degree of inhibition that exists in strong pepsin solutions, however, is so great that we found the results were more uniform if weaker solutions were employed, and all our later experiments have been made with 1 c.c. of a 1 per cent. solution of pepsin and 15 c.c. of the test substance.

The preparation of the tubes, however simple, is of some importance. The best results are obtained with fairly thick-walled glass tubes of an internal diameter of between 1 and 2 mm. These are filled with albumin, boiled for five minutes, and, while boiling, rolled continually. The ends are then closed with sealing wax. The variations in the reading of these tubes are so slight as to be of no importance.

Our results, of course, exhibit only one phase of the subject, and that is the effect of the test substances upon peptic digestion under apparently favorable conditions for its manifestation. In the stomach a number of factors are at work altering the composition of the gastric contents. These are: The ingestion of food, water, or saliva; the secretion of gastric juice; the secretion of mucus; osmosis; the discharge of the stomach contents through the pylorus; and, occasionally, the regurgitation of the duodenal contents into the stomach. In the case of saline solutions osmosis becomes a very important factor, and probably if strong they are rapidly diluted.

The technical difficulties of studying the effect of various substances upon the peptic activity within the stomach are very great; nevertheless, we have attempted to overcome them in a series of experiments which is still fragmentary and incomplete. It must not be forgotten, moreover, that in all these experiments the peptic activity varies not in accordance with a simple numerical proportion, but in proportion to the square root of the degree of concentration of the pepsin. This law was first determined by Schuetz, working with an exceedingly cumbersome method, and afterward restated by Borissow. Samojloff found that it did not hold good for strong solutions, either in artificial pepsin solutions or the actual gastric juice. In the course of our work we have noted some very good illustrations of this law.

TABLE I.

Pure	2.2	(4.84)
$^1/_2$	2.4	(5.76)
$^1/_4$	2.2	4.84
$^1/_8$	1.4	(1.96)
$^1/_{16}$	1.1	1.21
$^1/_{32}$	0.8	0.64
$^1/_{64}$	0.5	0.25

TABLE II.

Pure	4.9	24.01
$^1/_4$	4	16
$^1/_8$	2.7	7.29
$^1/_{16}$	1.9	3.61

In both tables it will also be observed that in the pure gastric contents inhibition was present. We shall discuss this more fully in a subsequent paragraph.

Schuetz's law is exceedingly interesting. Why pepsin should show the peculiar quality of diminishing in activity according to the square root of the degree of dilution—a quality, so far as we know, possessed only by it and a few other ferments—has never been clearly elucidated. An hypothesis that has occurred to us would serve in a measure to explain it, although, aside from the fact of being a satisfactory explanation, there is no other proof of its correctness. This is, that the peptic ferment consists of discrete particles. Either these particles are in continual activity and mutually interfere with one another in their movements, or else they radiate activity and the emanations from different particles interfere. To our minds the former hypothesis seems by far the more satisfactory, but in either case increase in the distance between the individual particles would cause an increase in their capacity for activity proportionate to the square of the distance, and inversely as the degree of concentration. Conversely, the effect of concentration would be to increase the peptic activity not directly, but in proportion to the square root of the degree of concentration. This accords with the results obtained by experiment. In high concentrations the inhibition is greater than the law would indicate, as is clearly shown in the following experiment:

Pepsin 1 per cent.	6.7	Pepsin $^1/_4$ per cent.	4.3
Pepsin $^1/_{16}$ per cent.	2.5		

If, in the gastric contents, the inhibition were of moderate degree the diminution of peptic activity in concentrated solutions would be sufficient explanation for it. Investigation, however, has shown that the inhibition is actually much greater than can possibly be explained by any concentration of the solution, being sometimes absolute, when pure gastric contents is tested, although in lower dilutions peptic activity is fairly strong. The following table of a series of cases taken in the order of their testing illustrates this fact very clearly. It has been arranged as follows: In the first column, the case number; in the second, the total acidity of the gastric contents under investigation; in the third, the peptic activity of the pure gastric contents; in the fourth, the peptic activity of the gastric contents diluted 1 in 4; in the fifth, the peptic activity of the gastric contents diluted 1 in 16. It did not seem necessary to insert the results of intervening dilutions, although they were usually made.

Case No.	T. A.	Pure.	$^1/_4$	$^1/_{16}$
173	96	8.6	6.6	5.5
66	52	0.0	5.2	2.8
168	43	4.1	5.5	3.2
188	145	5.6	7.1	5.9
118	90	7.8	7.5	4.3
151	150	2.2	2.2	1.1
134	60	8.9	8.2	5.4
151	150	8.3	7.9	5.8
151	150	3.7	5.4	3.7
134	60	0.7	3.5	3.2
155	106	7.4	7.3	5.3
139	58	0.0	...	2.2
134	90	0.8	4.5	4.8
120	136	2.4	...	1.9
124	40	3.2	2.4	1.2
118	72	5.2	3.1	1.7
H. C.	90	2.2	2.5	1.6
H. C.	67	4.9	4.0	1.9
H. C.	38	0.5	...	1.0
60	44	2.6	1.8	1.1
73	110	2.4	...	1.9
211	92	3.6	4.4	2.3
216	71	4.4	4.1	2.8

H. C. — hospital cases.

It will be seen that, according to Schuetz's law, the length of the column of albumin digested in the Mett tube should be, in the fourth column, twice that in the fifth, and in the third column, twice that of the fourth, or four times that of the fifth. In 5 cases the number in the fourth column is approximately twice as great as in the fifth, indicating that, in a dilution of 1 to 4, the inhibitory factors had been overcome. In no instance is the third column either twice as great as the fourth, or four times as great as the fifth. The average in the different columns is, for the third, 3.9 mm.; for the fourth, 4.8 mm., and for the fifth, 3.16 mm. In 2 cases the inhibition was complete, although in both digestive activity was present in higher dilutions (cases of achylia gastrica in which peptic activity was not manifest in the dilutions have been omitted). This table shows that the statement so frequently made, that in hyperacidity the peptic activity is at least normal, is entirely fallacious. In 1 of the negative cases the total acidity was 52, and in the other 58. In 2 cases, in which only a trace of digestion occurred in the pure gastric juice, the total acidity was 60 and 38. Inhibition was very pronounced in 1 case with a total acidity of 150, and in another with a total acidity of 145, and there was apparently an actual deficit in the peptic activity in the case with a total acidity of 136. These cases resemble in many respects achylia gastrica. It is obvious that in such cases the administration of alkalies alone can be of no benefit in restoring gastric digestion, but that the object of treatment should be to remove or diminish the inhibitory factors, because the results of the higher dilutions indicate that latent peptic activity exists in the undiluted gastric contents.

The literature upon the subject of inhibition by food and the products of digestion is not considerable. Nierenstein and Schiff believe that the carbohydrates have an active inhibitory power. Pawlow calls attention to the fact that the administration of oil greatly diminishes the secretion of the gastric juice, but he did not study its effects upon the digestive powers of the juice after secretion.

Pick is the only investigator who appears to have done much work upon this subject. His method differed in details from our own. He used a 2 per cent. solution of Merck's pepsin scales in $\frac{n}{20}$ HCl. Of this solution 20 c.c. were placed in a Stender dish with the Mett tubes, and the substance to be tested added in bulk. The whole was then placed in an incubator for twenty-four hours. Certain minor objections can be made to this technique. Excepting in a few cases it fails to determine the limits of inhibition. The substances added may alter the chemical reaction of the solution, or may act to a certain extent mechanically. The great advantage of his method is that it enabled him to make a large number of experiments within a comparatively short period of time, and therefore to cover more ground than could possibly have been done had he made more elaborate studies. He explains inhibition strictly according to the theory of ions. The electronegative ions inhibit peptic activity, and, therefore, those chemical compounds in which they predominate diminish proteolysis.

We used, wherever possible, solutions of known strength of the substances employed, and made a series of dilutions of these solutions, so that by comparing the final results with a series of controls, and repeating the experiments, it was possible to determine the points at which inhibition became manifest, and was absolute.

The effect of proteids was studied in various ways. 10 c.c. of a 5 per cent. solution of egg albumen were added to 5 c.c. of a 2 per cent. solution of pepsin, the object being to determine whether the digestion of egg albumen inhibited the digestion of the Mett tubes. There was no inhibition. A cylinder of egg albumen 1 cm. in diameter was then coagulated by prolonged boiling, and disks 4 mm. in thickness were cut from it and placed in the Stender dishes in place of the albumen solution, it being thought that perhaps there was not enough albumen in the other experiment. No inhibition, however, was observed. A similar experiment was then performed, but the Mett tubes were added only after the albumin had been digested for twelve hours, at the end of which period it was in fact entirely dissolved. Twelve hours later the average digestion was 3.3 mm. and of the control tubes 5.3 mm., indicating active inhibition as a result of the presence of the products of proteid digestion. In order to deal with a measurable solution we used Witte's peptone. The following series show its inhibitory power. In the first table 5 c.c. of a 1 per cent. pepsin solution was employed, and in the second table 5 c.c. of filtered gastric contents with a total acidity of 80.

Per cent.	Pepsin.	g. c.
10	0.0	0.0
5	0.0	0.0
$2\frac{1}{2}$	0.2	0.4
$1\frac{1}{4}$	2.4	0.7
$\frac{5}{8}$	2.7	0.9
$\frac{5}{16}$	3.0	0.8
Control	2.9	0.7

g. c. = gastric contents.

This shows that inhibition is absolute if the peptone or albumose present amounts to 5 per cent. by weight of the total, that it is still considerable for 2.5 per cent., but that it practically ceases about 1 per cent. Pick also found that Witte's peptone inhibited gastric digestion. To what extent this inhibitory activity is manifest in the stomach is difficult to determine. The methods for precipitating albumose carry down pepsin also, so that it is impossible to decide whether the removal of the peptone would restore peptic activity or not; and dialysis is too slow to be practicable. It seems difficult to understand how, in the two or three hours that the food remains in the stomach, enough albumose can be formed to produce a strong degree of inhibition, and there is no evidence to show that the inhibition is greater after a proteid meal than after an ordinary test breakfast of bread and water.

Somewhat akin to proteids is gelatin. We used the white transparent gelatin strips commonly employed in bacteriology. 1 c.c. of a 1 per cent. pepsin solution was employed as the digestant.

Per cent.	Pepsin.	g. c.	Per cent.	Pepsin.	g. c.
10	0.0	0.0	1/4	2.6	1.5
5	0.5	trace	5/8	2.5	1.7
2 1/2	3.0	1.2	Control	2.5	1.9

Presumably at a point slightly below 10 per cent. inhibition is absolute. It practically disappears at 2.5 per cent.

In testing carbohydrates we used first a solution of cane sugar dissolved in $\frac{n}{20}$ HCl. The digestant was 5 c.c. of a 2 per cent. solution of pepsin, in one series, and 5 c.c. of filtered gastric contents with a total acidity of 110, in the other series. Only one set of results in each series is given. Other series were practically the same.

Per cent.	Pepsin.	g. c.	Per cent.	Pepsin.	g. c.
10	1.0	0.0	5/8	2.2	2.5
5	1.7	2.7	5/16	3.1	...
2 1/2	2.2	2.4	Control	2.8	3.0
1 1/4	1.9	3.1			

In the pepsin group the digestive activity of one and one-quarter seems inaccurate. In three other series there was no diminution at this point. Practically, therefore, there is marked inhibition at 10 per cent., slight at 5 per cent., and little or none at 2.5 per cent.

In testing Merck's dextrose we used 5 c.c. of a 1 per cent. pepsin solution and 5 c.c. of filtered gastric contents with a total acidity of 80.

Per cent.	Pepsin.	g. c.	Per cent.	Pepsin.	g. c.
10	2.4	1.1	5/8	4.3	2.0
5	3.2	1.6	5/16	4.4	1.9
2 1/2	3.5	1.8	Control	4.1	2.1
1 1/4	4.0	2.0			

There was slight inhibitory activity above 2.5 per cent. In testing Merck's maltose 5 c.c. of a 1 per cent. pepsin solution was employed. The following is a typical series:

Per cent.		Per cent.	
40	0.0	1 1/4	2.5
10	2.4	5/8	2.4
5	2.4	Control	2.5
2 1/2	2.6		

Maltose, therefore, in dilutions of 10 per cent. or less has no inhibitory activity.

Lactose gave the following result with 1 c.c. of 1 per cent. pepsin solution:

Per cent.		Per cent.	
supersaturated	0.9	5	1.7
20	1.1	Control	1.8

On the whole the inhibitory activity of the carbohydrates is slight and not apparent in solutions of a strength likely to occur in the stomach contents.

The hydrocarbons were difficult to test. The method was that employed by Pick, olive oil being added to various solutions of pepsin and filtered gastric contents. Of course, the oil did not mix with the solution but remained floating on the surface, and, as was to be expected, no inhibition was manifest. As Pick has shown that acacia has a strong inhibitory action it was not feasible to make emulsions.

Alcohol was tested with 5 c.c. of a 1 per cent. pepsin solution. A number of series were made, the results being practically identical. As, however, the percentage limits in the different series were not always the same we have combined them.

Per cent.		Per cent.	
64	0.0	2	4.4
32	0.0	1	4.6
16	1.5	1/2	4.6
8	3.6	Control	4 3
4	4.2		

Complete inhibition, therefore, at 32 per cent.; slight at 16 per cent.; and little or no inhibition in mixtures lower than 8 per cent.

Conclusions of First Section. The results of the studies of the inhibition by food and the products of its digestion may be briefly summarized.

Albumin produces no inhibition during digestion.

Albumoses have a strong inhibitory effect. Under normal conditions this inhibition probably is not active in the stomach. Under abnormal conditions, particularly in cases of retention, it may be a serious factor.

Gelatin has a distinct inhibitory effect.

Of the sugars, maltose appears to exert the strongest inhibition, and lactose none at all. It is not probable that this inhibition ordinarily plays a very important role in gastric digestion.

Hydrocarbons apparently do not inhibit; at least, when they are added to the gastric contents.

In view of these results it seems reasonable to conclude that some factor other than the food interferes with the gastric digestion. That it cannot be deficient secretion of pepsin, seems clear from the fact that dilution of the gastric contents outside the body gets rid entirely of the inhibitory factor or factors. It is possible either that some factor is present which prevents activation of the pepsinogen or that some substance is lacking which is necessary to its activation. The question, however, is complicated. It is difficult to devise experiments which give decisive results; nevertheless, it seems capable of solution.

II.

The second part of our paper is concerned with the effect of various antiseptics employed in the preservation of foodstuffs. The controversy excited by the use of these substances has been exceedingly bitter, and, as is usually the case, experts have testified for both sides. In testing the effect of these preservatives reliance has hitherto been placed chiefly upon the effect produced by feeding foodstuffs treated with them to human beings or even to the lower animals for a considerable period of time. In some cases, very careful studies have also been made of the urine in order to determine whether they irritate the kidneys during elimination. Only a few attempts have been made to study the direct effect of those preservatives upon the digestive processes, and yet this would seem the most obvious thing to do, and the disturbance of digestion a most likely source of injury. Our investigations must not be considered as more than preliminary. We have endeavored merely to show what are the limits of the inhibitory activity of the preservatives when dissolved in artificial gastric juice or gastric contents. Of course, the same errors that apply to the study of inhibition upon the natural products of digestion apply in this case. Nevertheless, if a substance entirely prevents the proteolytic activity of the pepsin under the favorable conditions of an incubator and twenty-four hours' exposure in the Stender dish, it will certainly be effective in the stomach; and substances that in exceedingly moderate dilutions (such as salicylic acid and sulphite of sodium) interfere with digestion in the incubator, will also interfere with digestion in the stomach. It must not be forgotten that the mere inhibition or non-inhibition of peptic activity does not exhaust the capacity for harm of a particular preservative. Nor has the present series of investigations indicated what effect these substances have upon the proteolytic activity of the pancreatic secretion. The materials used have been, wherever possible, chemically pure.

As a preservative common salt is used principally for meats, fish, and butter. In saturated solution in $\frac{n}{20}$ HCl, using 1 c.c. of 1 per cent. pepsin or 1 c.c. of gastric contents as a digestant, a trace of digestion occurred when a saturated solution was diluted to one-eighth. The following results were obtained with known percentages:

1 : 20	Trace	1 : 160	1.7
1 : 40	0 8	Control	1.7
1 : 80	1.0		

Formaldehyde is used chiefly as a preservative for milk, or as a constituent in various preservative or embalming fluids. The dilutions were made with formalin, a 40 per cent. solution of formaldehyde, the active agent. Therefore, in order to obtain percentages of formaldehyde employed, the percentage or dilution that we give must be multiplied by 0.4. The following figures were obtained in different series of experiments:

1 : 10	0.0	1 : 80	1.7
1 : 20	0.0	1 : 160	3.0
1 : 40	0 6		

In this series the controls were unsatisfactory, and it merely indicates that absolute inhibition ceases between 1 to 20 and 1 to 40. In the second series the first column is with 5 c.c. of a 1 per cent. pepsin solution, and the second column is with 5 c.c. of filtered gastric contents of a total acidity of 75.

I.		II.	
1 : 100	2.0	1 : 100	0 9
1 : 200	2.6	1 : 200	1.2
1 : 400	2.7	1 : 400	1.2
1 : 800	2.9	1 : 800	1.3
1 : 1600	2.8	1 : 1600	1.5
Control	3.2	Control	1.5

Very doubtful inhibitory action in dilutions more than 1 to 100. In the beginning several series of tests were made in dilution of 1 to 1000 to 1 to 32,000, but no inhibitory effects were observed. We were rather surprised at the high concentration of formalin necessary to cause complete inhibition. These results agree with those of Bliss and Novy, who found that exposure to 4 per cent. formaldehyde did not destroy pepsin, but do not agree with those of Rideal and Fowlerton and Cassel, who found inhibitory activity. Starling believes that the formaldehyde prevents the digestion of fibrin that has been exposed to it, but has little effect upon the pepsin; perhaps an explanation of the different results.

Benzoic acid and benzoate of sodium are employed very extensively, particularly in preserves, pickles, catsup, etc. Benzoic acid dissolves very slightly in the HCl solution. In dilutions of 1 to 100 or higher the inhibition is absolute. In dilutions above 1 to 800 there appears to be no inhibition.

1 : 200	0 6	1 : 3200	4.0
1 : 400	1.5	1 : 6400	4.0
1 : 800	3.2	1 : 12800	4.1
1 : 1600	3.8		

Sodium benzoate in dilutions of 1 to 100 also causes complete inhibition. The following figures were obtained in a series of high dilutions, the digestant being 1 c.c. of 1 per cent. solution of pepsin.

1 : 200	1.6	1 : 800	2.4
1 : 400	2.0	Control	2.5

Boric acid is also extensively used. In supersaturated solutions it has no effect whatever upon the peptic digestion. This agrees with the results of Chittenden, Liebreich, and others.

Salicylic acid dissolves very slightly in the acid solution. As nearly as we could determine the saturated solution was about 1 to 500. The digestion was 5 c.c. of a 1 per cent. solution of pepsin. The results were as follows:

1 : 750	3.5	1 : 12000	5.0
1 : 1500	3.9	1 : 24000	6.1
1 : 3000	4.3	Control	7.3
1 : 6000	4.7		

With 1 c.c. of a 1 per cent. solution of pepsin the results were as follows:

1 : 200	0.8	1 : 20000	2.2
1 : 1000	2.0	1 : 40000	2.3
1 : 10000	2.1	Control	2.3

Sodium salicylate, when added to the HCl solution, is precipitated as crystals of salicylic acid, and the effects are probably the same. In all the dilutions which we employed there was total inhibition.

Sulphite of sodium is used in enormous quantities by butchers to restore the red color to the surface of meats that have been exposed to the air. In all dilutions lower than 1 to 100 it produced total inhibition. In the higher dilutions the results were as follows:

1 : 200	0.7	1 : 1600	3.4
1 : 400	2.0	1 : 3200	3.8
1 : 800	2.8	Control (average)	4.3

Even in dilutions as high as 1:1600, therefore, there is distinct trace of inhibition. It seems that this renders sodium sulphite particularly deleterious for employment upon meats.

Saltpetre (potassium nitrate) is another preservative frequently used. We did not determine the point at which it caused absolute inhibition. Solutions of 1 to 400 still showed slight inhibitory power.

Resorcin is not, as far as we know, used as a preservative, but was tested merely because of its extensive employment as an antifermentative and antiseptic in gastric conditions. The digestant was 1 c.c. of 1 per cent. solution of pepsin.

1 : 12	0.0	1 : 200	2.1
1 : 25	1.1	1 : 400	2.0
1 : 50	1.6	Control	2.2
1 : 100	1.6		

Inhibition ceases, therefore, if the dilution is in excess of 1 to 100.

Guaiacol carbonate is used for the same purpose as resorcin. It is almost completely insoluble in the HCl solution. The results were as follows:

Supersaturated solution	2.0	1/4 strength	2.3
Saturated solution filtered	2.2	1/8	2.3
1/2 strength	2.2	Control	2.2

Therefore, it has practically no inhibitory effect.

Creosote is also used as an antiseptic in gastric conditions and is one of the substances deposited in foods preserved by smoking. Creosote-water is prepared by mixing one part creosote to 100 parts water, agitating and filtering. The amount of creosote that goes into solution appears to be very small. We used $\frac{n}{20}$ HCl instead of water. The following figures were obtained, the digestant being 1 c.c. of a 1 per cent. pepsin solution:

Pure.	Trace.	1/8	1.6
1/2	0.6	1/16	1.9
1/4	1.0	Control	1.7

A few drops of creosote were added to 15 c.c. of the 1 per cent. solution of pepsin. There was a barely recognizable trace of digestion in the tubes.

Conclusions of Section II. Of the ordinary preservatives boric acid and borax may be regarded as without effect upon the peptic digestion.

Formaldehyde and alcohol appear to inhibit gastric digestion only when in high concentration.

Salicylic acid has a distinct inhibitory effect even in very high dilutions. This would seem to suggest that its reckless use may cause considerable impairment of nutrition.

Benzoic acid, sodium benzoate, and sodium sulphite inhibit very strongly gastric digestion.

Resorcin inhibits very slightly.

Creosote inhibits strongly, even in the high dilutions in which it is soluble in water or in acid solution.

On the other hand, carbonate of guaiacol, probably on account of its extreme insolubility in acid solutions, appears to have no inhibitory effect whatever.

III.

In the third section of the paper we report some experiments and investigations which had for their object the discovery of some method of diminishing the inhibitory activity of the gastric contents. It is certain that the inhibitory factor is destroyed by sufficient dilution, and that this dilution does not affect—excepting proportionately—peptic activity. Therefore, attempts were made to determine the effect of dilution upon the digestive power of the stomach contents. The method employed was that of Mathieu and Rémond for the determination of the total quantity of the gastric contents. After the withdrawal of a test meal 300 c.c. of water were introduced into the stomach and mixed as thoroughly as possible with the remaining stomach contents. It was then withdrawn. By estimating the total acidity of the test meal and of the diluted test meal it was possible to determine just what fraction of the latter consisted of stomach contents; that is to say, supposing the total acidity of the test meal was 100, and of the diluted test meal was 20, the diluted test meal must consist of four parts of water and one part stomach contents. Theoretically, then, if the stomach contents were diluted to such a point that inhibition was overcome (that is 1 in 16), it should possess a digestive power compared with that of the diluted test meal (further diluted 1 to 16) equivalent to the square root of five, or, to state it more generally, equivalent to the square root of the filtered test meal. In a series of these test meals we obtained the following results:

Case.	T. A.	Pure.	1/4	1/16
II. C.	90.0	2.2	2.5	1.6
Diluted	17.0	2.6	2.1	1.6
110	136.0	3.4	...	1.9
Diluted	42.0	1.4	2.5	1.1
124	90.0	0.8	4.8	
Diluted	19.0	0.6	3.4	
93	112.0	3.6	4.7	
Diluted	7.0	0.0	2.4	
151	70.0	6.7	4.5	
Diluted	5.5	1.0	2.5	
131	60.0	8.9	8.2	5.4
Diluted	8.6	2.3	4.6	3.3
97	66.0	9.0	7.9	5.2
Diluted	2.8	0.0	3.2	1.7
151	150.0	8.3	7.9	5.8
Diluted	13.5	4.1	4.4	3.0

In several cases the total acidity of the diluted portion was too low to permit digestion to proceed, and therefore the results were apparently negative. But in each instance it will be seen that when the acidity was restored by dilution (1 in 16) digestion became active. In no instance was the peptic activity of the diluted test meal (at a dilution of 1 in 16) as low as it should have been by the calculation, and in many instances it was almost as high as that of the test meal itself. There seems to be but two explanations: The introduction of the diluting fluid either stimulated the secretion of the pepsin, or else it converted some inactive form of pepsin into the active substance. In either case it caused a relatively great increase in the digestive power of the stomach contents. The therapeutic indication is not clear, but it would seem that the ingestion of large quantities of fluid in the late stage of digestion—that is one or two hours after taking a meal—might considerably enhance the digestive activity of the stomach. Of course, it might have, in addition, some injurious effect either upon the motility of the stomach or upon the digestive activity of the intestine. Neither of these points has been determined.

These experiments are interesting from another point of view, that is, from the standpoint of the doctrine of antipepsin. This subject has already been discussed, but these experiments show conclusively, we think, that some factor other than merely concentration is present diminishing the activity of the gastric contents. Otherwise the dilution of the original test meal should, at some stage, give results strictly proportionate to a similar dilution of the diluted test meal, and this appears not to be the case.

If the mere ingestion of a considerable quantity of water can stimulate the production of active pepsin, there may be some method of stimulating the secretion of active pepsin in the gastric juice in cases in which it is ordinarily deficient or absent. In 1888, Jaworski called attention to the fact that in cases of achylia gastrica, if a dilute solution of HCl was introduced into the stomach and allowed to remain for an interval of time, pepsin could be demonstrated in practically all cases. This statement we attempted to verify. As a general thing the method employed was to withdraw the test meal, then to introduce from 200 to 500 c.c. of $\frac{n}{20}$ HCl, which was allowed to remain from fifteen to thirty minutes. The peptic activity of the test meal and of the HCl solution was then tested. Altogether we have had four cases of achylia gastrica in which repeated examinations had failed to show any trace of peptic activity, either in the pure or diluted stomach contents, and one case of carcinoma of the stomach, confirmed by operation, in which there was a distinct trace of peptic digestion, both in the stomach contents and in the HCl dilution.

Case.	T. A.	Pure.	1/4	1/16
99	22	0.0	...	0.0
200 c.c. $\frac{n}{20}$ HCl fifteen minutes in stomach	...	1.3	1.3	1.9
H. C.	...	0.0	0.0	0.0
500 c.c. $\frac{n}{20}$ HCl twenty minutes in stomach	...	1.2	...	0.5
54	20	0.0	0.0	0.0
300 c.c. $\frac{n}{20}$ HCl fifteen minutes in stomach	50	0.0	1.5	...
H. C. (Carcinoma of stomach)	...	...	0.4	0.3
300 c.c. $\frac{n}{20}$ HCl fifteen minutes in stomach	...	3.7	2.8	1.4
53	...	0.0	0.0	0.0
300 c.c. $\frac{n}{20}$ HCl fifteen minutes in stomach	60	2.9	3.9	1.6

Whether the HCl serves merely to activate the pepsinogen, or also as a stimulant to its secretion, it is impossible to say. Presumably the latter, because in several of these cases the amount of acid present in the gastric contents was sufficient to make digestion possible had there been any proteolytic ferment. The method, unfortunately, does not seem to be of value in distinguishing between carcinoma of the stomach and non-malignant achylia gastrica, although our investigations are still too few to enable us to speak with any certainty on this point. It does yield, however, a very distinct therapeutic indication, that is, that in cases of achylia gastrica the HCl should be administered *before,* and not after, meals. It should also be administered in sufficient strength and bulk to insure active stimulation of the gastric mucous membrane. Of three cases of achylia, all of which have received it in this manner, two have been greatly benefited; the third had been brought in a fairly good condition by several years' treatment before it was employed.

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ACUTE UNILATERAL SEPTIC PYELONEPHRITIS.

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During the past seven or eight years, surgeons have been greatly interested in a class of cases which had been previously regarded as beyond the scope of operative treatment. Up to 1899 one finds a few scattered reports cases of acute unilateral pyelonephritis of the suppurative type which were operated upon. This condition was known as "surgical kidney" by our medical forefathers, because it so frequently followed operations or manipulations upon the urethra or the bladder, or was a result of infection of the viscera which could be almost invariably found upon the autopsy table. With the introduction of aseptic methods in our operations upon the lower urinary passages the number of cases derived from this source diminished. It was observed, however, as a not infrequent complication of the cystitis accompanying calculus, enlarged prostate, and gonorrhea, although even at this time it was thought to be so uniformly bilateral that any operative relief for its symptoms was contraindicated.

Gradually the attention of the profession was drawn to the fact that acute symptoms of a septic character accompanied by localized tenderness, pain, and often palpable enlargement of the kidney, were due not only to infection ascending from the lower urinary passages, but that there were many cases which could not be thus explained. It was found that the organisms in the remaining cases must undoubtedly have been carried to the kidney through the circulation as metastases from distant foci of suppuration. The discovery thus was made and confirmed by a large number of surgeons that these septic lesions of the kidney, whether urogenous or hematogenous, were not only unilateral in the early stages, but that the cases with the severest septic symptoms yielded promptly to nephrotomy or nephrectomy.

One of the earliest writers to call attention to these unilateral septic renal conditions was Goodhart, in 1868. He found[1] at 130 autopsies upon cases of "surgical kidney" that the lesion was present upon one side in 14.5 per cent. In an additional 71 cases collected from the literature he found 12 (16 per cent.) unilateral. In 1883, Singer reported a case of unilateral renal suppuration immediately following typhoid fever. The kidney had been severely injured eighteen years before. One of the most interesting contributions to the literature of the subject is that of Robert F. Weir.[2] The case was one in which he performed nephrectomy in a young man, aged twenty-five years, who had a severe urethrocystitis three months previously. The chief symptoms were fever of a remittent type (to 105°) and chills. The colon bacillus was found in pure culture in the extirpated kidney.

In 1895, Woodward[3] reported a case in which he had performed nephrectomy for a unilateral septic nephritis. The first case reported herewith was published in August, 1899.[4] It is superfluous to mention in detail the articles that have been written since 1899. Excellent reviews of the entire subject have been published by Klink,[5] in 1903, and by Wagner,[6] in 1904. We now know that cases of acute unilateral pyelonephritis with severe septic symptoms have a definite etiology and pathology, and that they can be recognized and are amenable to surgical treatment.

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[1] Guy's Hospital Reports, vol. xix.

[2] Medical Record, September 16, 1894.

[3] Annals of Surgery, 1895.

[4] Chicago Medical Recorder, August, 1899.

[5] Centralblatt der Mittheilungen aus den Grenzgebeiten der Medizin und Chirurgie, 1903.

[6] Schmdt's Jahrbucher, 1904.

ETIOLOGY. The causes are best divided into the two chief modes of infection, urogenous and hematogenous.

1. *Urogenous Infection.* The various modes of origin of this form are:

(*a*) Cystitis. This is one of the most frequent causes of an ascending infection. The cystitis is usually of a chronic type and associated with retention of urine. For this reason the renal condition is more frequent in males, since the causes leading to stagnation and retention of urine are more frequent, owing to the presence of an enlarged prostate or of a stricture of the urethra. The acute pyelonephritis of pregnancy and of the puerperium, which is often so difficult to recognize, can be explained upon the basis of the cystitis frequently present and so greatly favored by the stagnation incident to these conditions.

(*b*) Hydronephrosis and nephrolithiasis. Both of these renal conditions favor infection of the parenchyma, through the obstruction to the outflow of urine which they cause, added to the microorganisms so often present in these conditions.

(*c*) Unclean ureteral catheterization.

(*d*) Ureteral fistulæ. Here there is no resistance to the transmission of infection to the kidney.

2. *Hematogenous Infection.* This arises as follows:

(*a*) As a complication of subcutaneous injury of the kidney. Israel called attention to this form of acute pyelonephritis in his text-book, in 1901, and George E. Brewer[7] has recently contributed some valuable clinical and experimental observations, eleven cases of his own, and two of Joseph A. Blake, of acute pyelonephritis. Eleven of these occurred in women and two in men. There was a distinct history of injury in two and previous disease in one. In five cases there was recent history of some febrile disorder. In order to confirm his view that an injury to a kidney is frequently followed by acute unilateral septic infarcts he bruised the kidney and injected hypodermically varying amounts of cultures of organisms, such as Staphylococcus pyogenes aureus, Streptococcus pyogenes, Bacillus coli communis, Bacillus typhosus, and Pneumococcus. Of sixteen animals which, in addition to the inoculation, received an injury to one kidney, five showed no lesion, or only hyperemia and parenchymatous degeneration. Two of these animals died within twenty-four hours of acute septic intoxication. Of the remaining eleven, all developed distinct surgical lesions of the kidney. In eight the lesions were unilateral, and limited to the injured kidney. In three, the lesions were bilateral. In one of the bilateral cases the lesions were practically equal in extent and in severity. In the other two, the lesions in the uninjured kidney were mild in character, and undoubtedly would have subsided under favorable conditions. The renal lesions produced by these experiments were practically identical with those observed in my clinical cases. They showed considerable variation in number, extent of renal destruction, and degree of resulting toxemia. While these experiments have not been sufficiently numerous nor varied to investigate all of the causes which might direct a given blood infection toward one kidney, they demonstrate in a very conclusive manner that injury to a single kidney, whether produced by trauma, the presence of a foreign body in the pelvis, or an acute obstruction of the ureter, certainly acts as a strong predisposing factor in the evolution of a surgical lesion of that organ.

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[7] Surgery, Gynecology, and Obstetrics, May, 1905.

(*b*) As a result of catarrhal conditions of the intestine or as a complication of stagnation of the fecal current. Baginsky and Heubner have called attention to the frequent occurrence of septic renal conditions due to the colon bacillus, as a metastasis of these organisms, following gastrointestinal disturbances in children.

(*c*) After the acute infectious diseases septic emboli are very likely to lodge in healthy and in hydronephrotic kidneys.

(*d*) As a metastasis from furuncles, tooth infection, or any form of peripheral suppuration. Hessert has recently reported a case of acute pyelonephritis following a carbuncle on the neck, and Israel has observed several such cases.

(*e*) As a metastasis from the lower urinary passages or from the opposite kidney in consequence of transmission of the organisms through the circulation, and not by means of an intervening ascending infection. This form is very prone to develop after urethral or vesical manipulation.

PATHOLOGY. The most satisfactory division of the various conditions in which the kidney is found either at autopsy or during life is that given by Israel.[8]

1. *Ascending Catarrhal Pyelonephritis.* In these cases the mucosa of the pelvis is swollen and there is marked catarrhal inflammation of the collecting tubules of the kidney.

2. *Multiple Miliary Foci of Suppuration.* This is the most frequent and typical form. The kidneys removed in both of my cases belong to this group. The small foci of suppuration are irregularly scattered through the entire kidney, the cortex being especially involved. The foci, as in my second case, may be almost exclusively confined to one pole of the kidney. The cortical foci project usually above the level of the surrounding parenchyma. They are usually arranged in groups, each abscess being surrounded by a zone of intense hyperemia. On section one sees many streaks of purulent softening extending from the hilum to the cortex and radiating in a fan-like manner from the pelvis.

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[8] Nierenchirurgie, 1901.

3. *Hyperacute Form.* The course of the disease is so rapid that there is no time for the formation of foci. Examination of such kidneys shows only most intense congestion. At times there are some ecchymoses and turbidity. The entire cut surface is œdematous.

4. *A Few Large Abscesses.* This form is rare.

5. *Gangrene of the Pelvis and Gangrenous Foci in the Parenchyma.* This variety is very rare and its etiology unknown, but it is probably hematogenous.

A perinephritic abscess may result from the second, fourth, and fifth forms.

The organisms which may produce this condition are the Bacillus coli communis, Gonococcus, Staphylococcus aureus and albus, and Streptococcus pyogenes. According to Rovsing any urine-decomposing organism can cause the disease. At times it may occur during the course of a chronic pyelitis of hematogenous origin, or as a complication of tuberculosis of the kidney as a mixed infection.

Symptoms and Diagnosis. The symptoms vary according to the intensity of the infection. In general, one may speak of two classes of cases: The first class is the one in which the kidney is but little suspected to be the cause of the symptoms. In this variety the clinical picture is that of an acute, general septic intoxication. The principal symptoms are repeated chills, high temperature usually remittent, but sometimes continuous, rapid pulse, marked prostration, and nervous symptoms, such as restlessness, delirium, and stupor. There is usually marked leukocytosis. In all probability some cases which have been spoken of as cryptogenetic septicopyemia belong to this group, in which the general septic symptoms predominate. The local signs of a kidney infection are so slight that they are frequently overlooked, unless a very careful physical examination is made to exclude other causes of sepsis. These cases are often so acute in their onset (according to Brenner), and so rapidly progressive in their septic manifestations, that death may ensue before any renal or urinary changes show themselves. In the diagnosis the history of previous infection is of aid, as also that of some subcutaneous injury in the kidney region. The urine shows a few pus cells, or may contain nothing abnormal.

In the second class of cases the diagnosis is not so difficult, since the local signs predominate from the first and are accompanied by symptoms of septic intoxication. These local signs are: (*a*) Severe pain, most frequently located over the affected kidney. In the very acute cases this pain may not be so accurately localized, but referred only to the side of the abdomen corresponding to the affected kidney. When the right kidney is involved the differential diagnosis between this affection, appendicitis, gallstones, etc., is often very difficult. The onset is often as sudden as in any of the affections mentioned. (*b*) A second symptom of considerable importance is marked tenderness on pressure in the costovertebral angle of the affected side. (*c*) A third valuable sign, well-marked in the second of my cases, is rigidity of the abdominal muscles in the neighborhood of the affected kidney. In some cases one can palpate the kidney as enlarged and extremely tender. This sign was of great value in my second case as a differential diagnostic sign serving to exclude other acute suppurative diseases of the right side of the abdomen. The urinary symptoms are decrease in quantity of urine and the presence of pus in variable quantity. The general symptoms are quite characteristic, and are similar to those observed in the class of cases in which the general septic symptoms predominate over the local inflammatory ones. Chills occur at frequent but irregular intervals and are followed by a marked elevation of temperature. The fever is usually of a remittent type, but may be continuous.

The most characteristic symptoms are: Location of the pain, tenderness, and rigidity over the affected kidney, accompanied by pyuria, chills, fever, and remittent temperature. Such a unilateral septic process may occur in a kidney which has been the seat of a previous chronic condition, such as calculus or tuberculous pyelitis. One of the local signs to be especially mentioned is the presence of severe renal colics. These are due, as Israel has shown, to the extreme congestion of the kidney. The colics may simulate stone to such an extent that they are accompanied by anuria. The differentiation can readily be made from the graver septic symptoms present in an acute unilateral septic nephritis. If such a septic process occurs in a displaced kidney the differential diagnosis is almost impossible.

The following two cases will serve to illustrate this form of acute unilateral septic nephritis:

CASE I.—Mrs. B., aged twenty-six years, was admitted to St. Luke's Hospital June 4, 1898, on account of laceration of the perineum, cystitis, and suspected renal calculus. She stated that since the birth of her child (seven months previously) she had suffered from severe pain on urination, accompanied by the passage of calculi varying in size from a pea to a bean. Before the expulsion of these she had sharp pains in the region of both kidneys, with tenesmus and the passage of large clots of blood. There was intense burning and smarting on urination. The urine upon admission contained triple phosphates, pus, red blood corpuscles, and considerable albumin.

An exploratory nephrotomy performed in July, 1898, by Dr. W. H. Allport, resulted negatively. A little later the urethra was dilated, some small whitish particles were removed, and was followed by irrigation with silver nitrate. She was discharged on August 16, 1898, after her temperature had been normal for some time. The urine at this time contained a few pus cells; she still had incontinence, and occasionally passed pinhead-sized phosphatic calculi.

On August 24, 1898, she was readmitted, with a temperature of

105.4° F., and a pulse of 140. She stated that for the preceding four days she had suffered from excruciating pains in the region of the left kidney, which radiated down the ureter to the bladder and were accompanied by repeated chills and high fever. Examination revealed marked tenderness over the left kidney and very marked muscular rigidity in its vicinity. The urine contained a large number of pus corpuscles.

The diagnosis being made of pyonephrosis with probable renal calculus, I immediately performed an exploratory operation. The kidney was found adherent and greatly enlarged. Upon being brought into the abdominal wound, the many groups of miliary abscesses with hyperemic zones so characteristic of an acute pyelonephritis were seen scattered diffusely over the cortex. Nephrectomy was performed. Upon the following day the patient's temperature rose to 105°, and the urine was much clearer. The amount passed in twenty-four hours was 800 c.c. Upon the third day after operation she was removed to the isolated ward, on account of a faucial diphtheria; here she received 2000 units of antitoxin.

On her return to the surgical ward, on September 24, 1898, her temperature was normal; in twenty-four hours she passed 660 c.c. of urine containing pus and a few granular casts. The extirpated kidney showed many groups of miliary abscesses in the cortex, especially just beneath the capsule. On section yellowish streaks of purulent softening were seen extending from the apices of the pyramids toward the capsule. There were no calculi, but the pelvis was deeply congested and filled with pus.

The patient was operated upon after this twice during the next year: once for an ovarian cyst, and a second time for chronic recurring appendicitis. The latter operation was performed by myself. At this time (August, 1899), one year after the nephrectomy, her urine contained no pus; she suffered from incontinence during the day, but not at night. She passed from 46 to 48 ounces in twenty-four hours, and had gained twenty-two pounds in weight.

Case II.—Fred J. was seen in consultation with Dr. H. L. Baker, on October 27, 1905. He stated that about eighteen months previously he had been struck in the back with some degree of violence by a street car. He never felt well since that time, but had no definite symptoms, until about six weeks before my first seeing him. During this period he had severe pain in the back, fever, and chills. The cycles of recurrent chills and fever would last about a week; that is, he would have chills for a day, then high temperature, which gradually diminished, until at the end of four or five days he felt much improved. Then the chills and fever recurred. While in the hospital his temperature dropped from 104° to normal in three days. After being normal for twenty-four hours it suddenly rose to 104° again. There was marked rigidity of the abdominal muscles over the right kidney. The latter could be felt as enlarged and very tender on bimanual examination. The urine contained a large amount of pus and was alkaline in reaction.

The patient refused operation until November 2, 1905, when the temperature after having been normal for twenty-four hours rose to 104° and was again accompanied by very rapid pulse and great prostration. I made an incision over the right lumbar region parallel to the last rib. On exposing the kidney many adhesions were found. The organ itself was brought to the surface. The cortex of the upper half was seen to be studded with a very large number of miliary abscesses arranged in groups with a hyperemic zone. A few were also seen over the lower half. Nephrectomy was done.

The temperature became normal after the operation. The urine for first few days contained small amount of pus. Since then it has been clear. He began to resume active work as a painter about six weeks after the operation. He has gained thirty pounds in weight.

I believe that in the first case the infection was urogenous from a marked cystitis. In the second case a possible injury to the kidney preceded the urinary symptoms by over a year. There were no signs of cystitis before or after the operation, so that the infection may have been hematogenous.

Treatment. There are three distinct methods of treatment. The first is the medical, which may succeed in the milder cases: the administration of urinary antiseptics and the ingestion of large quantities of bland fluids, such as milk, water, etc. The temperature should be controlled, as far as possible, by hydrotherapeutic methods. The bladder should be irrigated at regular intervals with weak nitrate of silver. Ureteral catheterization should never be used because of the danger of infection.

The other two methods of treatment are of an operative nature and differ only in degree. The first is nephrotomy, and consists of exposing the kidney and opening the multiple abscesses with or without stripping off the capsule. There are many surgeons (like Israel, Brewer, Johnson, etc.) who claim that this method does not give as good results as nephrectomy. Some of the more radical advocates of nephrotomy even recommend the excision of the affected areas, if this be possible, in order to conserve as much of the kidney as can be saved. Nephrectomy has almost as many supporters as the more conservative operation. Johnson cured only one of seven patients by nephrotomy, the remainder being discharged with fistulæ. In a later series of five cases he performed nephrectomy three times successfully.

Which one of these two operations is to be performed in an individual case rests to a great extent on the experience of the operator. In my own two cases nephrectomy resulted favorably in both, but I believe that in any future case I should be inclined to give nephrotomy a trial, especially if the disease was not far advanced and the areas of suppuration were miliary in size or only slightly larger.

I have been able to collect from the literature 34 cases in which nephrotomy was performed with 28 recoveries (82 per cent.), and 27 cases of nephrectomy with 25 recoveries (92 per cent.). It is, of course, unfair to draw conclusions from these statistics, since the cases vary greatly in severity. If the condition occurs during pregnancy conservative operation is indicated. If the condition is a part of a general pyemia extirpation of the kidney is out of the question.

GASTRIC ULCER IN CHILDHOOD.

By Harry Adler, A.B., M.D.,
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The following case of hematemesis occurring in a child of eight years has been of considerable interest, on account of its apparent rarity, and caused me to study the subject of gastric ulcer in childhood:

B. L., white, female, has been attending school, and living under excellent conditions of sanitation and nutrition. The family history is negative. The child was breast-fed, and well nourished in infancy. At the age of two years she had a rather severe attack of acute gastritis attended with fever and persistent vomiting for several days. She also had German measles, but otherwise none of the infectious diseases of childhood. For several years she has been "taking cold" easily, having several attacks of bronchitis every winter.

Her present trouble is of about ten weeks' duration. She suffers with repeated attacks of pain in the epigastric region. The pain occurs in paroxysms both before and after meals. Often it begins before meals, is relieved for a short time by taking food, and then returns sharper than before. It persists then a varying time, from one to three hours. At times it comes on in the midst of a meal. Sometimes it disappears quite suddenly. While in my office on the first examination she suddenly became nauseated and vomited, without apparent effort, food streaked with blood. The vomited matter gave the reaction for free hydrochloric acid. One week later, during an attack of pain, she again became nauseated and vomited about an ounce of bright blood.

The patient is rather frail and pallid. She weighs 42 pounds. The lungs and heart are normal. There is marked tenderness to pressure in the epigastrium. The stomach area is normal on percussion. The liver dulness is normal. The spleen is not palpable. There is no tenderness over the rest of the abdomen.

Examination of the blood revealed hemoglobin, 72 per cent. (Gowers); red cells, 4,200,000; whites, 8200. The urine was normal. The stools after the first appearance of blood in the vomit showed blood with the turpentine-guaiac test. After the second appearance occult hemorrhage persisted for four days. Subsequent examinations failed to show its presence.

The patient was put to bed on a five weeks' ulcer cure. All food was withheld for thirty-six hours, after which diluted milk and bismuth were given. The stomach symptoms disappeared after the first three days, and remained absent during the entire course of treatment. Since then, a period of five months, the child has been free from symptoms and has gained steadily in weight.

From the symptoms, the attacks of gastralgia, epigastric tenderness, hematemesis, the presence of blood in the stools, and the result of treatment, the diagnosis of ulcer of the stomach is, I think, justifiable.

A study of the postmortem statistics shows that ulcer in children is absolutely and relatively of uncommon occurrence, as compared with its incidence in adults. Thus, in Welch's[1] collection of 607 cases of gastric ulcer, but one occurred in a child less than ten years of age. Lebert[2] found but one case in his series of 226. By Rehn[3] we are told that Rokitansky never encountered a case under fourteen years, and was himself, in 1873, able to collect but 9 cases in children. Fenwick[4] collected 18 cases, and Cutler[5] in 1904 added 11 cases. It must be remembered that these statistics have been gained largely from hospitals where postmortem examinations on children are comparatively infrequent. An examination of the statistics of children's hospitals shows a somewhat different result. Dusser[6] found ulcer in the stomach in 9 cases; and ulcer of the duodenum in 4. In 390 autopsies at the Babys' Hospital in New York, Martha Wollenstein[7] found 5 cases of gastric ulcer. Holt[8] reports 3 cases accompanying follicular gastritis and 5 accompanying general tuberculosis. In order to obtain data as to its occurrence clinically, besides consulting the text-books on diseases of children, I wrote to the leading pediatrists of this country, with the following results: Booker, Rotch, Griffith, Louis Starr, and Koplik have never seen a case that they recognized clinically. Koplik[9] reports one case seen at autopsy accompanying empyema, which he states represents his experience covering fully 300,000 cases of diseases in children.

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[1] Pepper's System of Medicine.

[2] Krankheiten d. Magens, 1878.

[3] Jahrb. f. Kinderheilkunde, 1873, p. 19.

[4] Disorders of Digestion in Infancy and Childhood, London, 1897.

[5] Transactions Amer. Gastroenterolog. Soc., 1904.

[6] Thèse de Paris, 1890. Cited by Holt, Diseases of Children.

[7] Cited from Osler, Practice of Medicine.

[8] Diseases of Children and Personal Communication.

[9] Ibid.

Jacobi,[10] on the other hand, states that it is not quite uncommon in children from seven to thirteen years. From the statistics of the children's hospital it appears that it occurs oftener than it is recognized clinically.

VARIETIES OF ULCERATION. Fenwick recognizes five varities of gastric ulcer: (1) Hemorrhagic erosions, (2) follicular ulceration, (3) tuberculous ulcers, (4) malignant ulcers, and (5) simple ulcer. The last may be either acute or chronic.

(1) *Hemorrhagic extravasations* into the gastric mucosa are frequently noted in children, as in adults. They are observed as the result of congestion of the vessels of the mucosa, secondary to uncompensated valvular disease or to dilated heart, complicating chronic pulmonary trouble, or more rarely in cirrhosis of the liver. They have also been seen in the hemorrhagic diseases, as purpura, scurvy, leukemia, hemophilia, and in jaundice. The hemorrhages vary from minute points, barely detectable by the naked eye, to irregular oval or linear areas. The smaller are limited to the mucosa, while the larger effusions may involve all the coats. This condition is of pathological rather than clinical interest. As will be seen later, Kundrat[11] considers the true ulcers to have their origin in the necrosis of the hemorrhagic areas.

(2) *Follicular ulceration* occurs for the most part in infants as the result of follicular gastritis. According to Fenwick: "Here and there small hemispherical masses about the size of a pin's head may be observed upon the surface of the mucous membrane. These appearances are due to inflammation of the solitary lymph glands situated in the wall of the stomach, and it is from the necrosis of these structures that the ulcers are produced. The swollen gland presses upon the gastric tubules which surround and cover it in, and gradually causes destruction of those in the immediate vicinity. A minute abscess is formed which opens on the surface." The disease seldom extends deeper than the submucous coat, but occasionally the muscular and the serous coats become involved.

According to Holt, these ulcers give rise to no symptoms other than those of the attendant gastritis, as a rule; he has, however, seen three fatal cases from hemorrhage occurring in young infants. The ulcers in these cases resembled those found in the intestines in follicular enteritis.

(3) *Tuberculous Ulcers.* Ulceration of the stomach due to tubercle is apparently less rare in children than in adults. Steiner and Neureutter met with 4 cases in 302 autopsies on tuberculous children. Widerhofer noted it twice in 418 cases, while Holt reports 5 cases in 119 autopsies in tuberculous children, but states that there is no positive evidence that the ulcers were the result of

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[10] Diseases of Children.

[11] Gerhardt's Handbuch der Kinderkrank., 1880.

tubercle. Together this is a total of 11 cases of ulcer in 839 cases, as against Durk's[12] findings of but 4 cases in 900 autopsies on tuberculous subjects.

(4) *Malignant ulcer,* or rather the occurrence of carcinoma on the basis of a gastric ulcer in childhood, is unknown.

(5) *True peptic ulcer* in children is either acute or chronic. Morphologically they do not differ essentially from those seen in adults. Kundrat states that recent ulcers differ from those in adults only in that they are not always round, varying from a hempseed to a lentil in size. Their pathogenesis is as little definitely understood as in adults, though certain of the causes which are given to explain their occurrence can hardly be accepted as operable in children; such as hyperacidity and degeneration of the bloodvessels of the mucosa. Kundrat considered them the result of hemorrhagic effusions in the mucosa, destroying its vitality and permitting autodigestion by the gastric juice. He says: "If, as is commonly considered, hemorrhagic infiltration is the cause of hemorrhagic erosions, then for ulcus rotundum the same cause must be accepted. The difference between them is quantitative only and not qualitative. In the former case only the mucosa is affected, while in the latter the gastric juice acts also upon the submucosa and muscular layers." In support of this view may be cited the fact that hemorrhagic erosions and true ulcers are often found together in the same stomach. Ewald[13] inclines to this for at least a portion of the cases. Gerhardt[14] furnishes examples of erosions changed into true ulcers.

It is difficult to explain the occurrence of ulcers in the first few days of life on any basis other than that during parturition a hemorrhage into the mucosa has taken place and been followed by autodigestion. An infectious origin in children is indicated by the occurrence of gastric ulcer in children dying of tuberculosis, typhoid fever, pneumonia, noma, diphtheria, and empyema (2 cases). Ewald looks upon the bacteria as causal in a mechanical rather than toxic manner. He cites the observations of Nauwerk, Letulle, Dieulafoy, Stokes, and Kraft, who found emboli consisting of masses of bacteria closing up the smallest vessels of the mucosa. These are supposed to give rise to circumscribed disturbances in the nutrition of the mucosa followed by necrosis.

One acute case was evidently of traumatic or infectious origin following severe burns of the arms and legs.

Site of the Ulceration. With the addition to Cutler's statistics of several recent cases in which histories could be obtained, acute ulcers are found as often at the cardia as at the pylorus. They were midway between the two openings in two, and generally distributed in two. In chronic cases there appears also to be no special predilection of site. Multiple ulcers widely separated are frequent.

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[12] Cited from Hemmeter's Diseases of the Stomach.

[13] Deutsche Klinik, p. 459.

[14] Virchow's Archiv., Band cxxvii, p. 85.

SYMPTOMS. These have been obtained from only some of the cases, the reports being inaccessible in some cases and deficient in others. In infants gastric symptoms are as a rule absent, indefinite, or masked by those of an infectious disease, a sudden hemorrhage, or pain from perforation, being the first evidence of the presence of ulcer. In a number of cases the ulceration was an unexpected pathological finding. In older children the symptoms are similar to those in adults, but seem to be milder. Excepting immediately preceding perforation, in but one case was the pain severe.

Pain and tenderness were noted in but 13 per cent. of the acute cases; but were present in over 60 per cent. of the chronic cases. The pain was usually not severe. In those cases in which the patients were able to impart information, it was localized in the epigastrium. In young children it seemed over the entire abdomen. In all cases it came on in paroxysms, occasionally before, but more usually after meals, and was relieved by vomiting. Intense pain was followed by hematemesis or perforation.

Vomiting. In the acute cases in infants. vomiting was usually subsequent to diarrhœa. In older children it followed epigastric pain and relieved the distress. In cases accompanying follicular gastritis, vomiting was persistent. Fenwick mentions the finding of minute pieces of mucous membrane in the vomit in cases of gastric erosions.

Hematemesis was noted in 40 per cent. of the cases in which histories were obtained. It was bright red and varied from a few streaks in the vomited material to a quart(?). In 4 acute cases it was the immediate cause of death; the stomachs were found filled with blood. In chronic cases the hematemesis was preceded by epigastric pain. In the acute, pain was often absent.

Symptoms of Indigestion. Anorexia, fulness, and distension, nausea, or heartburn were seldom complained of. Excepting acute cases, they were noted only in the chronic cases.

Stools. Diarrhœa was a prominent symptom, being present in almost one-half the acute cases. It usually preceded vomiting. Constipation was at times followed by diarrhœa. Melena was noted in the majority of cases of hematemesis. I have not been able to find a case in which it completely replaced hematemesis, although it is mentioned as a possibility, by Fenwick. Occult bleeding was, with the exception of my case, not noted.

Fever was noted in a considerable number of cases, running in infants as high as 106°. Cutler reports a temperature of 102° in a girl of six years and 101° in a child of eight.

Blood examinations were made in but 2 cases. Both were anemic. Hemoglobin, 75 per cent., and 60 to 85 per cent. In one the leukocyte count was normal; in the other, 25,000 to 27,000.

COMPLICATIONS. A striking feature of acute ulcer in children is the large percentage of *perforations*. In 18 cases in which autopsies were made perforation was found in 6, or 33 per cent. In 8 fatal cases of chronic ulcer it was present in 3, making a total of 9 cases of perforation in 26 fatal cases. The symptoms are sharp, sudden pain in the abdomen, vomiting and collapse. Death occurs within thirty-six hours. This complication is not infrequently the first indication of ulcer. In older children, symptoms of the complicating peritonitis are sometimes seen, as in adults; but in young children collapse with rapidly fatal termination is the rule. Bisset has recently reported a case of perforation in which the initial symptom was profuse hemorrhage. In 1 case, perforation of the diaphragm occurred with subsequent pyopneumothorax. In Chvotek's[15] case stenosis of the pylorus and dilatation of the stomach is recorded. The base of an ulcer had formed adhesions with the pancreas, and contracted narrowing the pylorus so that it would hardly admit the tip of the little finger. Krasnobayeff,[16] reports 3 cases of stricture of the pylorus in children.

Robinson[17] reports a case of chronic ulcer in a boy of fourteen years, in whom autopsy showed two strictures of the œsophagus with a diverticulum. The stomach was markedly contracted in size and the pylorus thickened and constricted. At the pylorus was a large ulcer almost encircling it. The mucosa was replaced by fibrous tissue to which the contracture of the stomach was probably due.

DIAGNOSIS. In those cases in early infancy in which the first symptoms are profuse hemorrhage or collapse from perforation, early recognition is obviously impossible. In older children the symptoms are usually so little distinctive, and the disease of such uncommon occurrence that the diagnosis must depend largely on the finding of blood in the vomit and stool. And here care must be taken to eliminate the many other causes of hematemesis and melena. Thus in Dusser's 24 cases of hemorrhage from the stomach and intestines, in only 13 was ulcer found. In infants the blood may rise from the gums, nose, throat, or lungs, and be swallowed, followed by vomiting. Blood may be sucked from the breasts of the mother, or, in neonats, have been swallowed during parturition. True hematemesis arises from a number of causes other than ulcer. It has been observed in purpura, hemophilia, scurvy, and similar diseases in which there is a tendency to bleeding from any of the tracts. Cirrhosis of the liver, a not very uncommon condition in children, may give rise to it. In malignant forms of the exanthemata, notably measles and variola, it is likely to occur. I have seen it in a case of influenza. Other causes are: corrosive poisoning, severe retching (as may occur in pertussis), and traumatism as due to swallowing of foreign bodies. The great majority of these can be eliminated with a little care. In cases of melena the elimination of other causes presents greater difficulties. It is present in practically all cases of hematemesis, either as visible or occult hemorrhage. The importance of its recognition lies in the possibility of it completely replacing the vomiting of blood. In addition to the causes already mentioned as producing hematemesis, the possibility of intestinal origin must be considered. These are ulceration of the bowel due to catarrhal enteritis or colitis, tuberculosis, and typhoid fever; further, intussusception, thrombosis of the mesenteric artery, intestinal parasites, rectal polyps, hemorrhoids, and straining at stool. In neonats, melena frequently occurs without discoverable cause.

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[15] Archiv. f. Kinderheilkunde, 1882, vol. iii, p. 267.

[16] Dietsh. Moskau, 1898, iii, p. 195.

[17] International Clinics, 1905, 15, s. iii, p. 119.

It is not unlikely that, with the attention now being devoted to the recognition of occult hemorrhage, curable ulcer of the stomach will be more frequently recognized in children suffering with chronic stomach disorders.

REVIEWS.

Diet and Dietetics. By A. Gautier, M.D., of Paris, France. Translated by A. J. Rice-Oxley, M.A., M.D. Pp. 552. London: Archibald Constable & Co. Philadelphia and London: J. B. Lippincott Co., 1906.

Diet in Health and Disease. By Julius Friedenwald, M.D., Clinical Professor of Diseases of the Stomach in the College of Physicians and Surgeons, Baltimore, and John Ruhräh, M.D., Clinical Professor of Diseases of Children in the College of Physicians and Surgeons, Baltimore. Second edition. Pp. 728. Philadelphia and London: W. B. Saunders Co., 1906.

Eating to Live, with Some Advice to the Gouty, the Rheumatic, and the Diabetic. By John Janvier Black, M.D., Fellow of the College of Physicians of Philadelphia. Pp. 412. Philadelphia and London: J. B. Lippincott Co., 1906.

The striking advances that have been made in our knowledge of the physiology of the alimentary canal in the past few years have stimulated general interest in dietetics and the treatment of disease by means of diet. This revival in the attention paid to alimentary physiology, coming as it does at a time when the general tendency of therapeutics is toward treatment by physiological measures rather than by drugs, has brought the whole subject of dietetics and alimentation into considerable prominence. Medical literature is as yet scantily supplied with works upon diet which are available for reference by the general practitioner, and it is with pleasure that we note the appearance of the volumes which are the subject of this review.

The books differ somewhat in their scope and character. Gautier's is characterized by its thorough presentation of the physiology of alimentation and the chemical and physical characteristics of different foods. Instead of furnishing arbitrary diet lists for each condition, he helps us to apply physiological principles to the diet of the individual patient. His consideration of diet in disease is brief and forms a comparatively inconspicuous portion of the book, but it is none the less admirable on this account. The first division of the book includes the physiology of digestion and of assimilation, as far as our limited knowledge will allow us to go. The question of the optimum amount of the different constituents of our food is considered in a manner that appears to us to be as satisfactory as is possible at this time. Gautier warns us against theoretical feeding, even if the principles upon which it is done appear perfectly rational. He says that the proteid and ternary optimums should be determined rather by the experience of the race than by chemical analysis—a statement which will meet the hearty support of most clinicians. The best proportions of the three main constituents of diet are most determined, according to Gautier, by studying the diet that accurately maintains a race at the highest point of intellectual and physical vigor. To do this, the analysis must include as large a number of individuals for as long a time as possible and must include all classes of society. Gautier does this by analyzing the diet of the three million inhabitants of Paris for ten years. For his conclusions we must refer the reader to the book.

Next, the physiology of digestion is considered, including a satisfactory review of Pawlow's work, especially the influence of the mind upon digestion. Then the principles of the realization of energy from food, experimental calormetry, and Atwater's respiratory chamber are fully described. The application of these principles in determining the indispensable minimum of proteid and ternary food is then considered. As we have inferred, Gautier opposes the idea that the indispensable minimum corresponds to the diet best suited for obtaining the highest development of the race. He shows quite clearly that such a minimum is fitted only for individuals in a condition of physical and intellectual inactivity. He then compares this diet with the ration chosen by workmen of different sorts in different conditions.

In the second part, the chemical and physical characteristics of different foods are well considered in a very practical manner. The food values of different articles of diet are clearly given, and the section is well suited for easy reference. A detailed description of this part of the book is impossible; but we note with pleasure the sections upon the value of broth as food, upon preserved foods, milk and its derivatives, eggs, cereals, and a full consideration of vegetable foods. Under the head of nervine aliments are included coffee, tea, and cocoa. Alcoholic beverages and alcoholism are given considerable space. Then follows a chapter upon condiments and inorganic foods, in which iron, the alkalies, phosphorus, etc., are considered. A series of chapters is devoted to drinking waters, and includes waters of the different spas, disease attributable to drinking water, and filtration. Part II closes with sections upon the preparation of food and arrangement of meals, both as to time and composition.

In the third main division are given the regimens best suited to various methods of living, classes of society, kinds of work, seasons, age, etc. A very moderate section upon vegetarianism points out the fallacies in the theories of the extreme supporters of such a dietary.

The diet in illness is then considered. As we have said, this does not form a prominent portion of the book, although its brevity detracts nothing from its importance. In it, Gautier briefly applies the principles that he has explained. Of course, unless one has been interested in what has gone before and has mastered the principles of alimentation, as far as we know them, this part of the book seems inadequate. It certainly contains no arbitrary diet list for different diseases, but in spite of this, indeed because of it, these chapters impress us as a very rational treatment of dietary in disease and a very fitting termination to a scientific and broad consideration of the whole subject of diet.

The translation by Dr. Rice-Oxley is decidedly above the average and is entirely adequate, with the exception of a few paragraphs.

In Friedenwald and Ruhräh's book the diet in disease occupies a much more prominent position. The principles upon which dietetics rest are not neglected, however, for the chapters upon the chemistry and physiology of digestion are very well done, although somewhat brief and condensed. The first edition of the book has already been reviewed in this department, so that a detailed reference to its contents is unnecessary; but for the purpose of comparison it may be well to speak in a general way of the authors' presentation of their subject. The chemical and physical properties of food, whether animal, vegetable, or inorganic, the consideration of water, mineral waters, coffee, tea, and alcoholic beverages, are all adequately considered. There are sections upon the preservation of food, artificial foods, preparation of food by cooking, infant feeding, and a diet for old age. This new edition also contains a more extended account of Prochowinck's diet for pregnancy complicated by contracted pelvis. The general consideration of dietetics closes with a concise, but very complete, account of rectal feeding.

The next 250 pages, comprising the bulk of the volume, consist of diet lists and regimens for different diseases. The authors do not confine themselves to the affections of the gastrointestinal tract, but consider the diet in practically all of the conditions treated by the internist and by the surgeon. The section upon disease, in which diet is a primary factor, includes diabetes, gout, obesity, under-nutrition, scurvy, and rheumatoid arthritis. We confess that we hardly see the propriety of including the last mentioned disease under this heading.

The last section of this book contains tables of army and navy rations, dietaries in public institutions, receipts, and rapid reference diet lists. These last dietaries are one of the best features of the book, because they seem to represent more of the authors' own experience and opinions than the more elaborate lists in the body of the volume. The book is an admirable one and much needed by the practitioner in America. The form in which the scientific principles of alimentation are given is more popular and more concise than in Gautier's work, and on this account will probably appeal more favorably to the general practitioner, and the form of dietaries given will make the book a popular one for reference with the clinician. It appears to us that the book is open to criticism in one particular, namely: that it is too evidently a work of compilation. While an admirable summary of the literature of dietetics, we feel that the book would have been more valuable still if the authors had included in it more of their own personality and had given us the advantage of their own judgment and experience—which they are so abundantly qualified to do.

Dr. Black's book is avowedly popular. It consists of a rather informal discussion of dietetics in general, the value of different foodstuffs, and the author's strong support of Chittenden's well-known advocacy of the reduction of the amount of proteids in our diet. Indeed, as the title implies, the real reason for the book's existence is to urge moderation in eating along the lines that Chittenden has indicated. Dr. Black has woven into his text much of his own views and reminiscences, and consequently much of his own personality, which adds materially to the interest and attractiveness of the monograph. The book contains a general discussion of digestion and assimilation, the value of different foods and dietaries for consumption, obesity, under-nutrition, gout, rheumatism, and diabetes, but beyond this no diet lists are given. J. D. S.

A TEXT-BOOK OF MATERIA MEDICA, THERAPEUTICS, AND PHARMACOLOGY. By GEORGE F. BUTLER, PH.G., M.D. Philadelphia and London: W. B. Saunders Co., 1906.

WE are informed that the present (fifth) edition has been entirely remodelled, rewritten, and reset in order to keep abreast of the remarkable activity in the study of pharmacology which has marked the past four years. One of the most striking changes to be noted in recent text-books on therapeutics concerns the classification of drugs. No system of classification hitherto devised is altogether satisfactory, and the plan adopted in the present work of grouping the various drugs according to the system of the body on which they chiefly exert their action has, at least, the advantage of being thoroughly practical. Some of the older subdivisions, such as antipyretics, analgesics, alteratives, astringents, and the like, have of course been retained, for obvious reasons. The question of classification, after all, has merely an academic interest and hardly deserves the enormous amount of thought and disquisition which has been expended upon it. Every practical requirement is satisfied if the book is provided with a table of contents, and a comprehensive general and clinical index, as is the case in the present instance.

In the introduction the author touches upon certain general questions that have an important bearing on the action of drugs and other remedial agents, and now form an integral part of our theories of disease and the process of recovery and return to normal condition. We say touched upon advisedly, as it seems to us that their treatment is too brief for a text-book on therapeutics. Where else indeed shall we look for satisfactory summaries of such questions as osmosis, electrolytic dissociation, and Ehrlich's side-chain theory of immunity? The literature on the subject consists chiefly of papers written for the investigator rather than for the general student, and requiring interpretation and even a certain amount of popularization before they can be assimilated and applied to practical medicine. This criticism might be thought unjust were it not that the author distinctly states in the preface that his book is intended essentially for students and practitioners, not for experimental workers in pharmacology. Nor do we agree with the author that much of the material which has been eliminated in the present edition from the chapter on serum-therapy belongs more properly to pathology. A thorough understanding of the theories of immunity is, in the present state of therapeutics, almost as essential to the practical doctor as to the laboratory worker, and the subject may very properly be included in text-books on practice and therapeutics.

On the other hand, matters of more immediate practical application, such as methods of administration, dosage, the secondary effects of drugs and the like receive full consideration in the introductory portion of the work. In the matter of dosage, unfortunately, we have not advanced beyond the stage of empiricism, and the practitioner is practically dependent on his own experience, and his knowledge of his patient's individual reaction to drugs in general, and to special medicines. The proper selection of the dose is usually more difficult than the choice of the drug itself, and this probably explains why the results obtained with the same agent differ so much in different hands.

The dose of digitalin is given as from $\frac{1}{100}$ to $\frac{1}{30}$ of a grain, although much higher doses are generally employed when a distinct effect is desired—as high as $\frac{1}{8}$ or even $\frac{1}{4}$ of a grain. It is also stated that digitalin is insoluble in water, whereas it can readily be dissolved in water for hypodermic administration. Aspirin, we are told, may be given in doses of 30 to 45 grains. The same dose is mentioned by Cushny in his last edition, so that there is authority for such heroic doses. Personally, we have seen distinct salicysm produced in an adult by 10 grains, four times a day, on the second day of administration, not to mention the fact that in such large doses the drug is a decided irritant to the stomach. Several of the newer drugs which have recently come into general use and have proved their efficacy are not considered worthy of mention. However, a certain degree of conservatism is quite proper in a systematic text-book, and it is undoubtedly difficult, at this early date, to give a sound opinion on remedial agents, which must still be regarded as in the experimental stage.

A dozen pages are devoted to organo-therapy and serum-therapy. In spite of all the work that has been done recently in these two fields of therapeutics there is virtually nothing new that is of any practical value to be recorded. Diphtheria antitoxin is still the one serum of which it may be said that it has become a permanent part of the Pharmacopœia, with the possible exception of Pasteur's virus for the cure of rabies, the importance of which is limited only by the comparative rarity of the disease.

The arrangement of the subject-matter and the paragraphing of the book are excellent, and the author is to be congratulated on his success in presenting everything that is essential in a concise, summarized form. In its new edition the book will maintain the place it has already achieved among the standard current text-books. The clinical index is a very valuable and convenient addition.

R. M. G.

UNTERSUCHUNGEN UEBER AMINOSAUREN, POLYPEPTIDE, UND PROTEINE (STUDIES OF AMINOACIDS, POLYPEPTIDS, AND PROTEINS). By EMIL FISCHER. Berlin: Julius Springer, 1906.

THE CHEMISTRY OF THE PROTEIDS. By GUSTAV MANN. Based on Professor Otto Cohnheim's *Chemie der Eiweisskörper*. London: Macmillan & Co., Limited, 1906.

THE book by Fischer is a collection of the papers that have been written by himself and his co-workers upon this subject since 1899, preceded by a discussion, eighty-three pages in length, of the reason that these studies were undertaken, and the purpose that has been followed in them, together with a consideration of the chief results that have been obtained. The papers themselves and Fischer's preliminary discussion are all technical, and will be uninteresting and incomprehensible to persons that have not some knowledge of chemistry. To physiological chemists, however, and to students of nutrition, they are of intense interest, since they furnish some of the most remarkable and important knowledge that has been contributed in many years. Our present knowledge of the nature of protein substances—that is, that they are constituted of groups of amino-acids bound together, in the case of the different proteins, in differing amounts—and the profound changes that this has made in our conceptions of digestion and nutrition in general are due, in large part, to the studies contained within this publication. In addition to this the work contains the studies on the formation of the polypeptids—substances that constitute the first approach to the synthetic production of protein. It was Fischer's reference to these substances in his paper before the German Chemical Society, the paper which opens this book, that led to such excited statements on the part of the lay press that he had practically made protoplasm and might be expected soon, perhaps, to make man—statements that he greatly deplores in a footnote, and that were clearly mere newspaper sensationalism. He expresses, on the contrary, in the original paper the attitude of the ideal scientist, when he states that it would be unfortunate if, by a mere lucky chance, someone should today light upon a method of artificially producing a true protein; for this would really contribute very little to chemistry or biology, and it would deprive scientists of the present fascinating opportunity to go on by slow and painful, but accurate, steps until, the whole matter having been carefully put together, protein will some day be produced in a manner that can be explained in each of its details.

As its title indicates, the book edited by Dr. Mann is, in most important particulars, practically an English edition of Cohnheim's well-known work. As the English editor explains, he takes the responsibility for this edition wholly to himself, because he has made certain modifications that are not in consonance with the views of Cohnheim, and thinks it more just to take this entirely upon his own shoulders. In many ways, however, the book has been put into a form quite different from Cohnheim's. On the whole, the author's reasons for not simply publishing a translation of Cohnheim's book, with notes by the editor, seem scarcely sufficiently forcible. He differs with Cohnheim chiefly in points of theory, and such differences anyone can excuse and can accept the theories of either at their proper value. The essential importance of the book lies in the fact that it is a very valuable discussion, which had not been attempted satisfactorily before Cohnheim did it, of the actual knowledge, rather than the theories, of the chemistry of the proteins. A very considerable part of the knowledge that is collected in the book is dependent upon the contributions of Fischer and his students, which have been reviewed under this same heading.

The book takes up the dissociation products, primary and secondary, of protein; the synthesis of albumins, a subject that is, as yet, of course, extremely incomplete; and the carbohydrate, sulphur, and other radicals. There is then a discussion of albumoses and peptones; of the salts of the albumins; of the special reactions, combinations, and oxidation-products; of the general physical properties of albumins according to Cohnheim and according to the author; and also of the classification of albumins according to each of them. There is then a discussion of the chemistry of the albumins proper, the globulins, the vegetable albumins, and the various other substances included under the term protein.

To the worker in physiological chemistry and in disorders of nutrition the book is of great value, and such workers are deeply indebted to Cohnheim for first collecting the information that it contains and to Mann for presenting it in English form.

D. L. E.

AUTOINTOXICATION INTESTINALE. By DR. A. COMBE. Paris: J. B. Baillière et Fils, 1907.

IN discussing the question of autointoxication from the digestive tract, two main courses are open: one, a course that was followed for example in a most interesting way by Weintraud in a short article in Lubarsch and Ostertag's "Ergebnisse," is to consider the whole subject from a critical standpoint, and admit nothing as wholly acceptable under this heading except those conditions that have, with a considerable degree of scientific accuracy, been shown to be the result of autointoxication from the alimentary tract; admitting, of course, at the same time, that there are suggestive reasons for thinking that other disorders may be due to this cause. The other course is to accept, as many authors have done with a lively enthusiasm, all sorts of conditions as due to this cause, providing that there is the slightest indication that the gastrointestinal tract shows abnormality and that treatment of such abnormality causes improvement in the symptoms; and providing, also, that other evident reasons for these symptoms are not found.

The latter course has been followed by Prof. Combe; and, indeed, in some instances he goes even further than this, practically attributing some conditions that are almost certainly due to disturbance of the thyroid, for example, or of some other internal organ, to intoxication from the digestive tract, which, he would have us believe, secondarily produces the disorder that is commonly considered to be the causative one. The reviewer cannot at all agree with the correctness of this attitude.

The book is written in a very interesting way, taking up the manner in which the protein molecule is broken down in digestion, the nutritive and toxic products of this process, and the experimental work that has been done in attempting to demonstrate the occurrence of intoxication in disorders of the digestive tract; then discussing at considerable length a large number of clinical conditions that the author attributes to intoxication from this cause; and reporting numerous studies of urine, etc., in support of his views. He finally devotes an extensive portion of the book to a detailed discussion of the dietetic and other treatment of such conditions.

This latter part of the book is the most valuable, and giving attention to the details recommended is likely to do good to many persons who have the various symptoms that the author describes; but enthusiasts about this matter overlook the fact that proper dietetic, hygienic, and other measures are likely to do good to persons with many disorders, whether their disturbance is primarily due to disease of the digestive tract, or to any one of a great number of other conditions, and whether it is toxic or nutritive. Consequently, the therapeutic test, while very valuable to the patient when successful, is not convincing evidence of the truth of a theory. One needs to accept some of the references in the work with caution, at least in the sense in which they are used; they have evidently been employed somewhat hastily to support the author's views and the form in which they are put does not always correspond with the actual views of the original authors. D. L. E.

LECTURES ON TROPICAL DISEASES. Being the Lane Lectures for 1905, delivered at the Cooper Medical College, San Francisco, Cal. By SIR PATRICK MANSON. Chicago: W. T. Keener & Co., 1905.

TROPICAL diseases in themselves are an interesting subject for study and an important one, at the present moment, for all Americans. In the Lane Lectures, which are the most delightful reading, the reasons for this importance are made perfectly clear, and the mode of transmission of certain tropical diseases from person to person, the spreading from country to country and continent to continent, is made easy of understanding. Besides these general considerations, the etiology, pathology, and symptomatology of several specific diseases are discussed in separate chapters: ankylostomiasis, dracontiasis, bilharziasis, trypanosomiasis, and several others. The author has been so closely associated with the study of these diseases that he speaks not only with authority but often from first experience. The book is altogether charming and one absorbs the really great amount of information which it contains in the pleasantest possible manner. W. T. L.

PROGRESS

OF

MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF

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The Diagnostic Value of Koch's Tuberculin.—ROTH-SCHULZ (*Beiträge zur Klin. der Tuberkulose*, 1906, vi, 167) gives the results of the use of tuberculin as a diagnostic agent in the Beelitz Sanatorium from 1902 to 1904. Over 1000 patients received the injections, nearly all being doubtful cases without lesions demonstrable by physical examination. The method employed is to administer as the initial doses 0.5 mg. of tuberculin after the temperature has been carefully observed for from four to seven days. Injections are undertaken only when the temperature is normal or shows a very slight elevation. If no reaction occurs after the first injection, in from two to four days, the same dose is usually repeated, particularly if any symptoms suggestive of a mild reaction have occurred with the first. If the temperature shows any irregularities, a longer period is allowed to elapse. Occasionally, the same small dose is repeated a third time, but, as a rule, at the next injection 1.25 mg. is given. If still no reaction occurs, this amount is occasionally repeated, but usually the dose is advanced to the maximum of 2.5 mg., which again is repeated once or even twice. A reaction is considered positive if the temperature rises 0.5° C. above the previous maximum, but the general and local phenomena are considered of great importance. The occurrence of rales where previously they were definitely absent or the occurrence of severe general symptoms are considered positive in spite of only a slight elevation of temperature. The importance of repeating the same dose of tuberculin is emphasized. Commonly a merely suggestive reaction upon the first administration is followed by severe symptoms after the second. This curious feature is analogous to the hypersensitiveness which sometimes develops during diphtheria and tetanus immunization and, indeed, may occur upon the administration of various sera. In Roth-Schulz's series of cases there is no relation between the sex, age, or general condition of the patient and the degree of reaction to the drug. The male patients react as a rule to smaller doses than the female but no factor is discovered to explain this. The duration of a lesion and its acuteness apparently bear no relation to the intensity of the reaction. The slight, closed, early cases seem to react more readily than others. As to the specificity of the reaction, we must depend chiefly upon the reports of veterinarians, and these are now almost unanimously agreed that when a definite reaction occurs tuberculosis must be present. The more carefully autopsies are performed the lower the percentage appears of reactions without demonstrable tuberculous lesions. The frequent occurrence of marked reactions in apparently normal adults and the absence of tuberculin sensitiveness in the newly born corresponds, too, very closely with our knowledge of the occurrence of tuberculosis in man. The fact that so many apparently normal adults do react positively to tuberculin is the one objection to be raised to its use as a diagnostic agent. It would be foolish to employ it indiscriminately and treat, in a sanatorium, everyone who reacts. It is merely a link in our chain of evidence and the careful consideration of the accompanying symptoms, which render the patient a suspect, are of the greatest importance for diagnosis. Marked reactions to small doses are very suggestive of an active lesion.

Clinical Contribution to the Etiology and Pathogenesis of Hepatic Cirrhosis.—Jagie (*Wien. klin. Woch.*, 1906, xix, 1058) reports a number of cases of cirrhosis of the liver which have been carefully observed during the past few years in the late Dr. Nothnagel's clinic. He points out the unsatisfactory classifications at present obtaining and insists upon the importance of discovering if possible the underlying cause of the condition. The anatomical classification, for instance, is a very unsatisfactory one, as alcohol, among other poisons, may cause two distinct types of the disease. In his first group of cases, numbering seventeen, the clinical picture is fairly uniform. All of them are marked alcoholics, and the initial symptoms of the disease those of alcoholic gastritis and enteritis. Jaundice occurs early in all, and is of a various and varying degree. One special symptom upon which Jagie lays great stress is the occurrence of attacks of pain in the hepatic area with tenderness over the liver. This he considers an important diagnostic feature. According to Naunyn, the attacks of pain and the jaundice are referable to an associated cholangitis. With every cirrhosis there is a certain degree of obstruction to the flow of bile, and this stasis must favor bacterial infection. Jagié points out that conversely infection from the catarrhal intestines may play an important etiological role in these cases. In the second group, numbering five cases, the importance of a chronic tuberculous lesion, as a causative factor, is strongly emphasized. A history of excessive alcoholic indulgence is absent; but all give a definite history of symptoms produced by tuberculosis, arrested apical lesions, pleurisy, protracted and severe bronchitis. The autopsy in all five cases, too, showed old pulmonary, glandular, or bone tuberculosis which had, in all probability, antedated the cirrhosis. The clinical picture is quite different from the alcoholic cases. The attacks of hepatic pain and the jaundice are absent. Indeed, the symptoms of any liver derangement are so slight as scarcely to draw attention to the organ. The importance of tuberculosis as an etiological factor in cirrhosis of the liver is further emphasized by certain animal experiments. Bensancon and Griffin, by the injection of pus from cold abscess, and Bartel by the injection of tubercle bacilli of low virulence, have observed in guinea-pigs, changes in the liver identical with cirrhosis as it occurs in man. Other authors have obtained similar results. Hansemann describes a form of spontaneous tuberculosis in guinea-pigs regularly associated with cirrhosis of the liver and often accompanied by attacks of jaundice.

Mechanism of Compensation in Tricuspid Insufficiency.—FRANKE (*Wien. klin. Woch.*, 1906, xix, 956). Many clinicians, considering the anatomical arrangement of the circulation, have contended that no satisfactory compensation of a tricuspid lesion can be established, as there is no driving force back of the lesion except the inefficient right auricle. As is shown clinically and experimentally, the left ventricle cannot overcome the deficiency as the right does in mitral lesions; in animals, after artificial damage to the tricuspid valves, the left ventricle is normal or a little atrophied. Franke cites cases to show that a certain degree of compensation does occur, that patients with serious tricuspid lesions may live comfortably for considerable periods of time, and that even after compensation has been broken, it may be satisfactorily re-established. He considers three factors as important in maintaining it. Hypertrophy of the right ventricle keeps up pressure in the pulmonary circulation and this pressure may be above normal, as is evidenced by the much accentuated second pulmonic sound. During diastole it exerts a suction action which assists the flow of blood from the auricle and veins. The elasticity and passive resistance of the auricle and large venous radicals prevent, to a certain degree, regurgitation. The contractile force of the hypertrophied right auricle, Franke thinks, is of small moment, as the presystolic wave formed in venous pulse tracings by its contraction is scarcely visible and certainly not accentuated above the normal. A very important feature, however, and a rather ingenious assumption is the position he gives the peripheral vessels in maintaining compensation. Hasebrock has drawn attention to what he terms the systole and the diastole of the peripheral vessels, and has pointed out its importance in sustaining the general circulation. Franke likens this action, which he thinks is accentuated by the passive congestion of tricuspid insufficiency, to the role the right ventricle plays in mitral lesions. This contractile power in the peripheral vessels, he believes, is most evident in the liver. The systolic regurgitant wave beating back into the veins finds its freest path along the hepatic vein, which is without valves. The greatest damming back, then, must occur in the liver, which is the meeting point of two venous waves coming in opposite directions; one the hepatic, the other the portal system. The pressure in the portal vein measures only from 7 to 13 mm. of mercury, while that in the hepatic, with an actively working hypertrophied right ventricle, must be decidedly higher. It is difficult to understand the absence of ascites in such cases unless there is some manner of regulation, and Franke ascribes such a self-regulation of the blood-flow to the liver itself. He assumes that, during the cardiac systole which is the diastole of the liver, blood is sucked in from the portal vein to be thrown with renewed force into the inferior vena cava by the succeeding contraction of the liver vessels and from here drawn into the right ventricle by its suction action.

The Intestinal Origin of Pulmonary Anthracosis.—SCHULTZE (*Münch. med. Woch.*, 1906, liii, 1702). The object of this article is to refute the conclusion of Calmette's students. This French school is accumulating more and more evidence in support of the intestinal origin of pulmonary tuberculosis, and recently van Steenberghe and Grysez have revived, from an experimental standpoint, Villoret's view of the intestinal origin of anthracosis. In their experiments on guinea-pigs and rabbits they mixed coal-dust and particles of India ink with the food, which, in some instances, they allowed the animals to eat, in others introduced directly into the stomach by means of a tube. At autopsy the pigment was found in the lungs and only in the lungs, the abdominal organs and mesenteric glands being free. The same result was obtained by intraperitoneal injections. In from twenty-four to forty-eight hours after an intraperitoneal injection of India ink they were able to demonstrate an anthracosis of the lungs and the tracheobronchial glands, while all other organs showed no pigmentation. Schultze, in repeating these experiments, comes to different results. After the intraperitoneal injections he finds a pigmentation of the lungs, but these are neither the only organs affected nor do they show any special predisposition toward its accumulation, for there is more pigment in the spleen and the liver than in the lungs. In his feeding experiments he obtained the same results as van Steenberghe and Grysez, the pigment being recovered only in the lungs, but his interpretation is different. He feels convinced that in feeding experiments, even when undertaken with the aid of a tube, it is impossible to exclude inhalation, and, indeed, this is how he explains its deposit in the lungs in these experiments. The fact that it is found only there, he believes, is almost convincing evidence as the distribution is so different when it is spread by the blood-stream. In a rabbit with a gastrostomy fistula he introduced pigments into the stomach over a period of two months and at section was able to find no deposit in the lungs.

Estimation of the Functional Capacity of the Heart.—FRITZ LEVY (*Ztschr. f. klin. Med.*, 1906, lx, 64). Ketzenstein has shown experimentally that when a large section of the arterial tree is suddenly cut off there is a rise of blood-pressure, and he has applied this principle to the functional study of the heart in man. The method is to determine the pulse-rate and the systolic blood-pressure in the brachial arteries and then compress by digital pressure the femoral arteries. Normally, a rise of blood-pressure of from 5 to 15 mm. of mercury occurs, while the pulse-rate remains constant or falls a little. Under certain conditions this rise is absent or there may even be a fall in the pressure associated with an increase in pulse-rate, a condition which Ketzenstein interprets as evidence of cardiac insufficiency. In cases of marked hypertrophy the rise is considerably more than 15 mm. Levy has applied this method extensively and details his results. In most normal hearts he finds a rise of pressure does follow femoral compression, the rise rapidly subsiding when pressure is removed. The patients showing no rise were nervous, easily excited women in whom the method cannot be employed. Well-compensated valvular lesions display the normal rise, as do also neurasthenics with subjective heart symptoms. In cases of marked hypertrophy a rise of 15 mm. is the minimum to be considered normal. Anything less than this is classed as an insufficiency, and Levy quotes cases to illustrate the point. In wasting diseases and acute infections a fall, instead of a rise, is frequently observed and is held to indicate a serious loss of power in the heart. A similar fall was observed in some cases of valvular heart lesion, and the course of the disease confirmed the bad prognosis indicated by the method. The results in convalescence from acute infectious diseases are ranked as of a high order of importance and seem to furnish some idea of the condition of the heart. A convalescent typhoid patient, in spite of a drop of 10 mm. of mercury and a rise in rate of from 8 to 10 beats, was allowed out of bed because he felt so well, but was immediately taken with severe prostration. A patient with scarlet fever had a similar attack after leaving bed while the blood-pressure reaction was a falling one. As improvement occurs the rise after compression becomes steadily greater and greater. A case of mitral insufficiency showed a similar steady inclination to rise during recovery from broken compensation. Levy thinks that one of the greatest values of the method is in following injury done the heart by an acute infectious disease and in enabling us to gauge the length of time necessary to spare the heart any overexertion.

The Effects of Severe Muscular Exertion on the Circulatory Apparatus, the Kidneys, and the Nervous System.—Baldes, Heichelheim, and Metzger (*Münch. med. Woch.*, 1906, liii, 1863) have made examinations of the participants in a 100 kilometer walking race before and after the contest. Those engaged in the race were young men between the age of seventeen and twenty-seven years. Of the twelve examined, all were normal except one whose urine contained a trace of albumin without formed elements in the sediment. Immediately upon finishing, the urine of all showed albumin varying in amount from a slight trace to 0.5 gram per liter by Esbach's method. In four cases the presence of blood was determined by chemical reaction. The specific gravity of all cases was very high. Casts and epithelial cells were found in nine cases and red blood corpuscles in the four whose urine showed the presence of blood chemically. By percussion the heart outlines in all cases were so appreciably increased that the authors, taking into account the possibility of error by this method, are still convinced that there must have been an acute dilatation. There was a much lower blood-pressure reading after than before the race. The pulse-rate was much increased, but by palpation no special changes in quality could be discovered. There was an average loss of four pounds in weight during the contest. The general condition of the contestants was good. There was no unusual dyspnœa, no palpitation, no cyanosis. In one there occurred a short fainting attack. Several days after the contest the authors were able to examine three of the participants, and the abnormal conditions noted immediately after the race had completely disappeared.

SURGERY.

UNDER THE CHARGE OF

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Concerning Reported Recurrences after Prostatectomy for Simple Hypertrophy.—ANDRE (*Annales des Maladies des Organes Genito-Urinaires*, July 1, 1906) says that he formerly believed with others that such recurrences could occur, but that now he does not think this occurs after the surgeon has removed the enlarged prostate as completely as possible by the recent methods. He refers to a case in which, at first, he believed that reproduction of prostatic tissue took place, but the later evolution of the affection showed that cancer was present. Without doubt a partial prostatectomy can be followed by a true recurrence, but when a complete operation is so followed, it is probable that a cancerous prostate and not a simple hypertrophy was present. The diagnosis between the two conditions is often difficult. During the operation on a second case he was able to recognize the presence of cancer.

The Surgical Treatment of Subclavian Aneurysms.—SAVARIAUD (*Revue de Chirurgie*, July 10, 1906) states that aneurysms of the subclavian artery should be divided into two groups those external to the scalenus, and those which are external and internal to the scalenus. The first group is essentially surgical. They may be treated by extirpation, or by ligature, which should be placed as far as possible in advance of the aneurysm. The combination of the two methods may be employed, the Anel ligation to be followed by the extirpation if this seems possible. For the second group the ligature of Anel is too dangerous, while the Brasdor or distal ligature gives a mortality almost nil, and the later results frequently are remarkable.

Gastrocutaneous Fistulæ, Consecutive to Gastric Ulcer.—PATEL and LARICHE (*Revue de Chirurgie*, July 10, 1906) say that this condition is relatively rare. It results from a perigastric focus of suppuration opening on the external surface, spontaneously or from an incision. It may result without the formation of an abscess that is clinically appreciable. It is then due to a gradual invasion from mild infections. The point of election is the upper part of the stomach, which is least movable and can the more easily adhere to the abdominal wall. Once established their characteristics vary according to their location. If they are high near the heart, the discharge is intermittent; if low near the pylorus, the discharge is constant. In the former a good general condition is maintained for a long time; in the latter, on the contrary, it rapidly becomes bad. In the former medical treatment may be tried, the edges of the wound being left to cleanse themselves, and operation being done only if a cure does not result. The direct methods of operation, by excision of the ulcer and suturing the opening in the stomach, are to be preferred to the indirect method of gastroenterostomy. In the case of a fistula situated low, one should attempt its closure at once. The best method is to close the fistula by direct suture, although this is not always followed by success.

Pain in the Bladder in Women.—Rochet (*Annales des Maladies des Organes Genito-Urinaires*, July 15, 1906) calls attention to frequent attacks of pain in the bladder, rebellious to treatment, which cannot easily be attributed to the ordinary causes of cystitis, as gonorrhea, tuberculosis, etc. In the great majority of cases the urine is disturbed and the bladder infected. In certain cases, however, the urine though examined with care, does not show apparent trouble and is almost clear. In general, blood is rarely observed in the urine. Upon vaginal examination and the passage of a urethral instrument, pain is not experienced until the neck of the bladder is reached. A careful examination of the bladder, the kidney, and the urine will sometimes be necessary to eliminate a beginning tuberculous cystitis. Tabes must sometimes be eliminated also. The pain may be due to gonorrhœal or puerperal infections, of long standing, which have been overlooked.

The Open-wound Treatment of Skin Grafting.—Goldmann (*Zentralb. f. Chirurgie*, July 21, 1906) says that he has had excellent results with the open-wound treatment of skin grafted fresh and granulated surfaces. Organic fixation of the transplanted flap, as has been frequently shown, occurs after twelve to twenty-four hours by means of the abundant newly formed fibroblasts and capillaries. Previous to this it is more or less mechanical from the plasma. The chief object should be to secure this fixation during the first hours, and for this purpose, the open-wound treatment will be most effective by drying the cementing substance. According to Goldmann's experience, after the grafts have once taken hold, the kind of dressing is not important. Either a moist or oily dressing may be employed to dissolve the crusts or scabs, so that the grafts may not be injured. The essential advantage of Weischer's method is not due to the periodical irrigation with salt solution to prevent coagulation of the secretion at the edges of the grafts, but to the accumulation of a cementing substance from the removal of the superficial granulations. Goldmann says that the vesical formation does not occur between the granulating surface and the grafts, but between the stratum Malpighi and the mucosum of the epidermis, and is an expression of the defective nourishment of the skin. As soon as the grafts are sufficiently nourished it disappears.

Exposure of the Heart in Wounds of this Organ.—Wilms (*Zentralb. f. Chirurgie*, July 28, 1906) says that the flaps ordinarily employed are suitable for the suturing of wounds of the anterior surface of the heart, but make a suture of the posterior surface difficult. In operating on a case with an anterior wound of entrance and a posterior wound of exit he found it necessary to resect the sternum before he could suture the posterior wound. He thinks that a long intercostal incision, as employed by Mikulicz and Sauerbruch, would, after opening the pericardium, give a free exposure for attacking the posterior surface of the heart. It should be placed in the fourth or fifth interspace, according to the situation of the wound. Division of the fourth or fifth rib would add much to the extent of the exposure. Moreover, the duration of the operation would be considerably shortened.

A Contribution to the Study of Cancer of the Rectum.—Martin Du Pan (*Revue de Chirurgie,* August 10, 1906) concludes that sedentary occupations, habitual constipation, hemorrhoids, and traumatisms have not the importance generally accorded them in the etiology of cancer of the rectum. Its development is much more rapid before forty years of age than in advanced age. Its insidious origin is one of the principal obstacles to the improvement of the final results from operation. Early diagnosis is rare. It will be more common, and the operative treatment more beneficial, when physicians generally follow the practice of digital examination of the rectum in all troubles of defecation. In the author's statistics the average duration of life in most patients not operated on is twelve months; in those subjected to radical operation it is fifty-six months. The height of the cancer, the presence of indurated glands, adhesions to the prostate or vagina are not contraindications to extirpation of the rectum. Immobility alone is an absolute contraindication. A preliminary colostomy should be avoided if possible, and should be employed only in cases of ileus so grave as to put the life of the patient in immediate danger. The coccygeal operation of Kocher consists of a posterior incision in the median line, with resection of the coccyx. It is the method of choice for the removal of tumors of the ampulla and superior part of the rectum. The best treatment of the upper cut end of the intestine is to fix it at the anus, with or without invagination in the inferior end deprived of its mucus membrane. The circular suture of the two cut ends should be abandoned, because of the great danger of infection. The peritoneum, however, should be sutured as exactly as possible, after fixation of the intestine, and drainage should be avoided. When infection appears the wound should be opened at once and irrigated. The author's mortality was 14.58 per cent., and was favored by the use of ether instead of chloroform. Local recurrences operated on survived in 39.7 per cent. of the cases. Careful watching of the patient after operation is, therefore, important to detect recurrences, as they rapidly become inoperable. Immediate hemostasis, as complete as possible, and the avoidance of denuding the neighboring bone, are important, in order to avoid vascular metastases and cancerous adhesion to the bone.

Wound of Thoracic Duct; Ligature; Recovery.—Fullerton (*Brit, Med. Jour.,* June 16, 1906) says that during the removal of tuberculous glands on the left side of the neck the thoracic duct was ruptured. On sponging away the escaping opalescent fluid the open mouth of the ruptured duct was seen and immediately caught with an artery clip. The duct on the proximal side of the ligature immediately became distended and seemed ready to burst. The distal end was found but did not allow the escape of fluid. It opened by three smaller vessels into the internal jugular vein. It was ligated, the wound closed with drainage, and during the latter dressings no fluid escaped. The patient recovered.

From a study of the recorded cases the dangers would appear to be: (1) Entrance of air into the veins; (2) rupture of the distended duct on the proximal side of the ligature; (3) inanition from draining away of the chyle; (4) possible dissemination of tubercle from entrance of the organisms into the torn duct. The treatment consists in occlusion of the torn duct by ligature, clamps, or packing, and keeping the patient quiet on a low diet to limit the pressure in the obstructed duct.

Concerning Ligation of the Renal Artery and Vein.—JUNGANO (*Annales des Maladies Organes Genito-Urinaires*, June 15, 1906) says that from the result of experiments on three rabbits and two dogs, he concludes that complete arrest of the circulation in the renal artery and vein so interferes with the nutrition and function of the kidney that a nephrectomy should be done. The collateral circulation is not sufficient. The renal epithelium undergoes necrosis and is replaced by cicatricial tissue, the necrosed material being absorbed and producing a toxic effect on the organism. This noxious effect is increased when the organism is in a condition of lessened resistance, as from a lesion of the renal vessels, from the hemorrhage which ensues. Besides, the injuring instrument may introduce infection, which in the area of degeneration finds a propitious field for development.

Etiology and Treatment of Oblique Inguinal Hernia.—MURRAY (*Brit. Med. Jour.*, June 16, 1906) concludes that the main factor in the causation of the hernia is the presence of a preformed sac and a patent funicular process. Whether the bowel will or will not enter this funicular process depends, primarily, on the size of the opening at the internal ring; and secondarily, on the strength of the muscles guarding it. The only satisfactory treatment is by operation, and the earlier the better. The essential point is to obliterate so completely the sac as to render the peritoneal surface free from any dimpling or depression. The divided aponeurosis of the external oblique should be subsequently united by overlapping, for the inguinal canal is thus narrowed, lengthened, and its original obliquity restored.

The Treatment of Cancer of the Stomach.—ROBSON (*Lancet*, August 18, 1906) says that there were 19,607 deaths from cancer of the stomach in England and Wales during the years 1901-4. 5 per cent. ran their course without local signs. Exploratory incision is the only reliable diagnostic resource in a considerable proportion of the cases. He advised that it should be done in a suspected case, if repeated examinations of test meals show diminished digestion and a diminution of free hydrochloric acid and the presence of lactic acid. In 59.3 per cent. of gastroenterostomies for later cancer, the long history of painful dyspepsia suggested the possibility of ulcer preceding the malignant disease. This is an additional incentive to the earlier surgical treatment of rebellious ulcers. Inflammatory masses about an old ulcer may so continue to obstruct the pylorus as to cause death. A gastroenterostomy sometimes causes a cure. Gastrostomy and jejunostomy for irremovable growth at the cardiac end of the stomach, in a fair proportion of cases, leads to great prolongation of life, while in thirty cases operated on by Robson there was only 5 per cent. mortality.

The good results from gastroenterostomy in irremovable pyloric growths are well established, and the presence of a manifest tumor does not mean that it is too late to perform a radical operation. The disease may involve a great part of all of the stomach, so that it is irremovable, and gastrectomy is impracticable. Robson has performed jejunostomy with advantage in two such cases. The patients were relieved of pain; one lived three months and the other twelve months. The immediate risks of partial gastrectomy as calculated from a considerable series of cases are between 14 and 16 per cent. Of twenty-seven cases of total gastrectomy, collected from all sources by Peterson, ten patients died, a mortality of 30 per cent.

The thorough removal of the disease may bring about as much relief to the patient as does the operation for the removal of cancer of the breast, uterus and other organs of the body, and in some cases a complete cure may follow.

Successful Treatment of a Case of Thoracic Aneurysm, Threatening to Rupture Externally.—Young (*Lancet*, September 22, 1906) reports a very interesting cure by what was practically the Tufnell treatment. It consisted of as nearly absolute rest as possible, restricted diet for a week and later an ordinary fish diet, no stimulation, and potassium iodide, grains 10 three times a day. The dose was quickly and steadily increased so that by the end of the third week 60 grains was being taken three times a day, with no ill effects at any time. As a local application to the swelling, collodion was painted all over the surface, every night and morning. Instead of continuing his previous downward progress, he commenced to improve from almost the commencement of the treatment. At the end of three weeks the patient said he had not been so well for four years. In about six weeks he was discharged apparently cured.

THERAPEUTICS.

UNDER THE CHARGE OF

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Calomel in Enteric Fever.—C. B. Ker (*Edin. Med. Jour.*, 1906, No. 1, p. 29), after discussing the use of various antiseptics in typhoid fever, lauds calomel as being superior to them all, particularly when employed in connection with intestinal irrigations. He denominates his treatment "antisepsis by elimination," and has used it in 758 cases of enteric fever. The treatment consists in administering aperient doses of calomel (3 grains) every second or third day, supplemented by hot water irrigations of the colon. The diet is chiefly milk, and water-drinking is encouraged. The advantages of the method are that it produces no pain and does not cause diarrhœa. The percentage of perforations was under 2 per cent., a rather low figure; hemorrhage occurred in 8.04 per cent., which seems large, but the loss of blood in many of the instances was slight. Marked meteorism is a contraindication to the use of calomel, but this manifestation may be caused to disappear by giving a small irrigation, and calomel may be allowed after twenty-four hours, unless blood appears as the result of the enema. Mercurialism was never noted. The death-rate of the patients treated as above was 9.36 per cent., but in calculating these figures no deductions are made of cases dying within forty-eight hours after admission. The author concludes with the statements that the calomel treatment is not unsafe, and ulcerative accidents are not increased. He makes the claim that the treatment leaves the patient free to deal with the enteric poison and relieves him from septic absorption; the bowel is kept clean, meteorism is reduced, and diuresis is assisted. The suggestion is made that this method in connection with the use of a specific serum, when such is obtainable, would be the most rational to employ in enteric fever.

Leukocytogenous Serum in Surgical Infections.—PAUL DELBET (*Revue de Thérapeutique*, 1906, No. 11, p. 384) has employed the horse serum of Petit with considerable success in various surgical infections. The serum may be used either in fluid form or as a powder; it is applied locally to the lesion itself, or in its immediate neighborhood. It may be injected into the tissues or applied as a wet dressing upon gauze. The action of the serum is both local and general; under its employment the toxic symptoms become less marked, the temperature falls, and the pulse-rate diminishes. Locally, after from four to six hours, the wound becomes more moist and in the severe instances the pus becomes more abundant. No untoward symptoms have been noted. In puerperal infection the author packs the uterus with sterile gauze impregnated with the serum, and in the case cited rapid amelioration followed the procedure. Previous to the packing the septic contents of the uterus was removed by curettage. In four instances of appendicitis of severe grade success followed the use of the serum. In a case of postoperative peritonitis and in an instance of sloughing hemorrhoids the treatment failed.

The Effect of Potassium Iodide in Experimental Arteriosclerosis Due to Adrenalin Inoculations.—W. T. CUMMINS and P. S. STOUT (*University of Pennsylvania Medical Bulletin*, 1906, No. 5, p. 101) after observing a series of experiments in rabbits state tentatively as a preliminary report that potassium iodide may be effectual in preventing the development of arteriosclerosis due to adrenalin inoculations. It seems probable that the salt has no absorptive properties, after the arteriosclerosis has appeared. Further research is being carried on by the authors.

The Treatment of Aneurysm by Relaxation of Arterial Tension.—H. HUCHARD (*Journal des praticiens*, 1906, No. 20, p. 307)) reports three instances of aneurysm which he has followed, two for eight years and one for four years, in two of which permanent cure has resulted from treatment calculated to reduce vascular tension below the normal. The treatment consists in keeping the patient at rest in bed and in prescribing a diet from which soups containing an excess of fat, meats, especially those cooked rare, game, fish, cheese, salted foods, tea, coffee, spirits, heavy beers, and an excess of wine are eliminated. Tobacco is also forbidden. Drugs, such as trinitrine, tetranitrol, and sodium nitrite, were administered. The author considers that the iodides have been overrated in this connection. In syphilitic aneurysms mercurial injections are dangerous on account of their liability to affect the kidneys and, as a consequence, to cause increased arterial tension. The milk diet in connection with theobromine, which assists in eliminating vasoconstrictor poisons, is very helpful in reducing vascular tension.

Antithyreoidin in Exophthalmic Goitre and Kindred Affections.—H. L. Elsner and J. R. Wiseman,(*New York State Journal of Medicine*, 1906, No. 6, p. 235) state that they consider that antithyreoidin is a remedy which is effectual for the control of the annoying symptoms of typical and atypical cases of exophthalmic goitre. The greatest improvement is found in the relief of the tachycardia, precordial distress, and tremor. This improvement is hastened by rest in bed and diet regulation. These latter without the administration of antithyreoidin are much less effective. Improvement in one or more symptoms is likely to follow in from three to seven days after the beginning of treatment, but if no amelioration appears after three or four weeks the chances are against the occurrence of relief. The treatment should be continued for from four to eight weeks at a time, at intervals of two or three months, after subjective symptoms have disappeared. Patients without marked goitre, with slight exophthalmos, and the von Graefe symptom have been most benefited. The exophthalmos is the most rebellious of the symptoms. Nervous symptoms usually yield as the heart becomes slower. Most of the patients treated increased in weight, and when the antithyreoidin is stopped feel the lack of it greatly. The treatment produces no ill effects and cardiac dilatation and hypertrophy do not contraindicate its employment. The serum is administered night and morning in doses of from 15 to 30 minims, but in mild instances of the disease the initial dose is from 10 to 15 minims. It may be given in water, raspberry syrup, elixir of orange-peel, or a mild wine. If no benefit accrues from the above doses these may be increased to from 40 to 60 minims. Experience argues against permanence of cure in chronic cases.

Hypodermic Administration of Iron and Arsenic in the Anemia of Pretuberculous Conditions.—B. R. Shurly (*Jour. Amer. Med. Assoc.*, 1906, No. 24, p. 1833) believes that hypodermic medication with iron and arsenic, together with strychnine and the hypophosphites, offers a prompt and powerful reconstructive adjunct to the pure air, good food, and sensible hygiene that are essentials in pretuberculous conditions. The green ammoniated iron citrate can be introduced into the system without danger in doses of from $\frac{3}{4}$ to $1\frac{1}{2}$ grains, while sodium arsenate is given in doses of from 1/60 to 1/30 of a grain. The injections of solutions of these drugs are given deeply into the muscles of the buttocks or back. Only slight pain attends the procedure and a general feeling of well-being follows the treatment. A full dose of the iron within five minutes causes a feeling of tension in the head, tingling sensations, and a flushing of the face. Doses larger than 1½ grains may cause nausea or vomiting. The hypodermic method of administration obviates injury to the stomach or teeth and does not cause gastric disturbance or constipation. The treatment is contraindicated in fever or acute hemorrhage. Daily injections should be followed by a 5 to 10 per cent. increase in the hemoglobin at the end of the first week.

Nitrogen Injections in Tuberculosis.—L. Brauer (*Deut. med. Woch.*, 1906, No. 17, p. 652) reports an instance of tuberculosis which was characterized by hectic temperature and extensive lesions, and in which nitrogen was injected upon four occasions, in quantity about three pints at a time. After the first injection the fever disappeared for about three weeks and after the subsequent injections similar results were noted, but they were less lasting. The collapse of the lung caused neither dyspnœa nor cyanosis and the patient's general condition exhibited improvement. The technique of the injection was as follows: The site chosen was in the seventh intercostal space between the posterior axillary and the scapular lines at a point where the pleura seemed to be normal and the lung little affected. Under local anesthesia an incision about an inch long was made down to the parietal pleura. The latter was punctured by a trocar and cannula, through which the gas was injected, having previously been passed through 1 to 1000 mercury bichloride solution. The wound was then sutured. The nitrogen is but slowly absorbed and the good effect of the treatment is produced by compressing the lung, lessening its congestion, and putting it at rest. The compression is evidenced by an increase in the expectoration following the procedure. It may be that a condition of venous stasis is established which acts upon the infectious process.

OBSTETRICS.

UNDER THE CHARGE OF

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Abdominal Section during Pregnancy for Carcinoma of the Ovary and for Ovarian Tumor with Twisted Pedicle.—Schauta (*Zentralb. f. Gyn.*, No. 25, 1906) reports abdominal sections during pregnancy for ovarian tumor with twisted pedicle. The first case was that of a multipara, whose labors had been normal. At the fourth month of pregnancy she came under observation complaining of pain in the right lower portion of the abdomen. The pain suggested disease of the appendix. Under anesthesia a tumor the size of a fist could be found which was movable. Abdominal section was made with a right lateral incision, and a parovarian cyst with twisted pedicle was removed. The pregnancy went on undisturbed. The second patient was in the fourth month of pregnancy when she was taken with severe pain in the right lower portion of the abdomen. This was thought to be appendicitis. The patient remained in bed eight days, when she was apparently better. Pregnancy proceeded and was terminated by labor. Four weeks after labor the patient was examined, and she was found to have a tumor the size of a fist, directed toward the left side of the abdomen. This tumor did not seem to be connected with the uterus. At operation an ovarian tumor was found adherent, and with a very long thin twisted pedicle. The appendix was adherent to the tumor and very little altered in its substance.

Schauta also reports a case of carcinoma of both ovaries complicating pregnancy. The patient was five and one-half months advanced, and complained of various indefinite pains in the abdomen, with fever and enlargement of the abdomen. This rapidly increased and on examination the uterus could scarcely be felt. On opening the abdomen a large quantity of ascitic fluid was removed, and five months' pregnancy was found present, and in addition a tumor of each ovary as large as a fist. These were found to be carcinoma with dissemination in the peritoneum of Douglas' cul-de-sac. The broad ligaments were removed as extensively as possible with the tumor. On the next day following operation the patient aborted.

Schauta also reports an interesting case of a woman who had had a tumor on the abdominal wall near the left flank, which was removed during the first month of a preceding pregnancy. From this she made a good recovery. In the seventh month of her fourth pregnancy a tumor appeared in the scar of the former operation. Through friction with the clothing, bleeding occurred from the tumor. Upon examination it was found to be as large as a child's head and involving only the abdominal covering. At operation the tissues were extensively removed, when the tumor was found attached to the fascia and attacking the peritoneum at only one point. It was a fibrosarcoma. The wound healed by granulation, as so much tissue was removed that the edges could not be brought together.

The Prevention of Difficult Labor.—Herman (*British Medical Journal*, June 30, 1906) contributes a paper upon this subject in which he calls attention to fatalities in labor following septic infection and disproportion in size between mother and child. Death from malpresentation and hemorrhage are largely avoidable. Death from eclampsia or rupture of the uterus in easy labor, or from acute yellow atrophy of the liver, cannot be foreseen and cannot be prevented. Accidents from disproportion between mother and child may be anticipated by measuring the mother's pelvis, and also by subjecting her to vaginal examination in order that tumors of the pelvis may be detected. Herman would not empty the uterus in cases in which the pelvis is so small that a living child cannot be born, but would let the mother go to term and perform, if necessary, Cesarean section. Pregnant patients should be examined after the seventh month, and if a malpresentation be found, attempts should be made to remedy it. When the back is posterior, the child can usually be turned by manipulation. If the child is obliquely, the head can usually be brought over the pelvic brim. In breech presentation it is often difficult to turn the fœtus about. Premature labor is advised in cases of moderate disproportion, the method being the use of a bougie, followed if necessary by dilatation of the cervix with solid dilators and the introduction of De Ribe's bag. When the pains are weak and inefficient they should be helped by the careful use of forceps. For a patient who has become exhausted from obstructed labor craniotomy is the proper treatment and not Cesarean section; the latter should be done early and not upon an exhausted patient. Hemorrhage from separation of the placenta, if threatening, Herman believes should be treated by Cesarean section. In placenta prævia the use of the dilating bag is recommended. Postpartum hemorrhage is usually caused by exhaustion, and especially by using forceps, without regard to contractions of the uterus. Efforts at delivery should always be made while the uterus is contracting. If the uterus is imperfectly emptied, postpartum hemorrhage may also be expected.

Pemphigus of the Newborn.—Koblanck (*Zeit. f. Geb. u. Gyn.*, 1906, Band lvii, H. 3) reports a case of congenital pemphigus, the mother a multipara, who had during pregnancy a moderate catarrh of the bladder and vulva. Labor began with discharge of amniotic liquid, and sixteen hours afterward a puny child was born. No vaginal examination was made during labor. Immediately after birth on the skin of the buttocks and upper arm were found pemphigus vesicles. The palms of the hands and the soles of the feet were free. Gradually bluish-red blotches appeared on the face and cranium. Syphilis could be excluded and the case was considered one of intrauterine infection originating in the external genital organs. The vesicles healed in a few days and mother and child remained in good condition. To determine the cause of the infection Koblanck examined the contents of the vesicles of fourteen children, and of two mothers who became infected in the mammary glands while nursing the children. One case gave no cultures whatever, probably because the vesicles were found in the stage of drying. In nine cases, one of which was maternal. Staphylococcus aureus only was found. In the remaining cases the germ found resembled Staphylococcus aureus. The infection probably occurs from mechanical pressure on the delicate skin of the child, but this may also happen in a grown person, as seen in cases in which the mother's breast becames infected by nursing the child. Experimental inoculation is usually made by incising the superficial layers of the skin.

A Fifth Case of Inversion of the Uterus Successfully Treated by Application of De Ribe's Bag Inflated with Air.—Pinard (*Annales de Gynecologie*, May, 1906) reports his fifth case of inversion of the uterus, successfully treated by De Ribe's bag. The patient was a multipara who had a normal pregnancy. Labor was terminated by an easy application of the forceps. The entire labor lasted twenty hours, the actual period of expulsion seven hours. Traction upon the os was made to deliver the placenta, when the uterus inverted. There was no hemorrhage, and the placenta, attached to the fundus, was readily removed together with some clots, but as the membranes were attached at the internal os, it was necessary to tear through the placenta to deliver the membranes completely. The uterus was readily reduced, the patient suffering very little shock. Four days afterward the uterus again became inverted after the patient had emptied the urinary bladder. Efforts at reduction by hand failed, whereupon De Ribe's bag was introduced and inflated with air. Its pressure did not cause pain, and within the twenty-four hours following, the uterus returned to its normal position. As a rule, in using the bag in this manner it should not be allowed to remain longer than eight hours.

Cesarean Section for Threatened Rupture of the Uterus.—Potocki (*Annales de Gynecologie*, May, 1906) reports the case of a patient, a multipara at term, who had been in labor twelve hours without the engagement of the head. Mother and child were in good condition. The pelvis was symmetrically contracted, the anteroposterior diameter measuring 9.8 cm. The type of pelvis was rachitic. When dilatation was complete the membranes were ruptured, amniotic liquid was slightly discolored. The anterior parietal bone presented, but the head still remained immovable. Engagement failed, although means were taken to relieve the patient's suffering. Her pains, however, became so severe as to demand prompt relief. The distension of the lower uterine segment was found to be excessive and uterine rupture was threatened; it was accordingly determined to deliver the child by Cesarean section. On opening the abdomen and the uterus the tissues were engorged and infiltrated with serum, giving evidence of severe birth pressure. A well-developed male child was delivered; the uterus packed with sterile gauze, which was carried through the cervix into the vagina, and the womb was closed by three rows of catgut stitches. Mother and child made good recoveries. The child's head was above the average in size and very well ossified.

Gonorrhœa in the Puerperal Period.—Mayer (*Monatschr. f. Geb. u. Gyn.*, 1906, Band xxiii, Heft 6) reviews the literature of this subject, and concludes that puerperal gonorrhœa may be a dangerous disorder. It can cause high fever and severe general infection with chills. The general picture may be that of systemic septic infection of severe grade. We cannot definitely distinguish how much of this condition is caused by toxins and not by bacteria. In severe cases with high fever we must suppose that mixed infection with streptococci and staphylococci is present in addition to gonorrhœa. Great variations in temperature, alternating between high fever and periods free from fever, are more characteristic of gonorrhœal infection than of sepsis. The statement sometimes made that fever developing late in the puerperal period is characteristic of gonorrhœa cannot be maintained. As regards the effect upon the children, in six children born of a gonorrhœal mother, four would be feeble or deficient at birth, while the other two would have less than usual power of resistance.

Gangrene of the Puerperal Uterus.—Schmidlechner (*Archiv. f. Gyn.*, 1906, Band lxxviii, Heft 3) reports the case of a patient who had had four normal labors, and who, during the fifth pregnancy, had considerable hemorrhage followed by an offensive discharge. The patient was thought to have had an abortion and was brought to the hospital. On examination a mass of decomposed tissue was found in the cervix. On further examination the uterus was as large as a fist, somewhat sensitive and anteflexed, and its wall very thin. Great care was taken to avoid rupturing the uterus, and a portion of the mass in the cervix was removed for examination. It was found to consist of necrotic uterine tissue, fibrin, disorganized and unrecognizable cells, blood pigment, and pus cells. The patient grew worse, although on examining the blood no pathological organisms of an infectious character could be found. She was thought to have chorio-epithelioma or gangrene of the uterus. Under anesthesia the uterus was removed through the vagina; the right ovary was also removed, and the stumps of the broad ligaments brought into the vaginal wound, and packing of iodoform gauze inserted. On examining the uterus its muscle was found to be a dirty grayish brown and highly disintegrated. In several places the uterine wall was almost perforated. On microscopic examination the tissues were found in a condition of cellular necrosis. The patient made a speedy and an uninterrupted recovery.

GYNECOLOGY.

UNDER THE CHARGE OF

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Dermoid Cysts of the Ovary.—Schottlaender (*Archiv. f. Gynäkologie* 1906, Band lxxviii, Heft 1) found in the walls of these cysts numerous cavities lined, and often filled, with epitheloid and giant cells, which represent dilated lymphvessels due to the irritation caused by the invasion into them of sebaceous material from the skin. This cell proliferation may be so marked as to justify the diagnosis of lymphangioma or endothelioma. The fact that these spaces are lined with lutein membrane does not prove that dermoids are developed from the ovum. According to Schottlaender they are secondary cysts; whether they all develop in this way, or are due to fetal inclusion, is not yet certain.

The Physiology of Uterine Contraction.—Kurdinowski (*Archiv. f. Gynäkologie*, 1906, Band lxxviii, Heft 1) from experiments on rabbits finds that cold excites contraction more than heat. Cold water acts more promptly in cases of uterine hemorrhage and a less quantity is required; nor is an intrauterine injection required, since the mere contact of cold with the portio is enough to excite general contractions. Long-continued mechanical irritation, as by rubber bags, leads to atony of the uterine muscle. Weak stimuli are better, such as electricity.

Intestinal Fistulæ following Laparotomy.—Osterloh (*Zentralbl. f. Gynäkologie*, 1906, No. 34) reported ten cases of intestinal fistulæ following laparotomy, with eight recoveries. In every instance the fistula healed spontaneously. In the discussion of the paper Leopold reported three cases, two terminating fatally; Peters two cases, in which the fistula closed without operation. The latter thought that in these cases the prime factors in the causation of the fistula was a peculiarly soft condition of the intestinal wall in a given case, especially when it was adjacent to a purulent collection.

Abdominal Myomectomy.—Bonier (Thèse de Lyons; Abstract in *Zentralbl. f. Gynäkologie*, 1906, No. 34) thinks that the ideal operation is removal of the fibroid alone, whenever this is possible, the disadvantages of total hysterectomy being the premature menopause and atrophy of the vagina. The younger the patient the more conservative should be the operation, although it may involve greater risk and difficulty. Bonier's own mortality in myomectomy is 3.3 per cent. The ultimate results were satisfactory. The uterus generally undergoes atrophy and the climacteric is normally established. Recurrence of the fibroid is rare.

Tuberculosis of the Cervix Uteri.—Lannes-Dehore (Thèse de Lyons; Abstract in *Zentralbl. f. Gynäkologie*, 1906, No. 34) finds that tuberculous disease of the cervix is most common between the ages of twenty and thirty years, occurs equally in nulliparæ and in those who have borne children, and that the infection may be either primary or secondary. Leucorrhœa is always present, and often amenorrhœa, dysmenorrhœa, and metrorrhagia. Fever was noted in only one case observed by Lannes-Dehore. Clinically three varieties may be presented—the miliary, ulcerating, and proliferating. The treatment is surgical unless advanced tuberculosis is present in other organs.

Ovarian Tissue in Dermoid Cysts.—Gentili (*Archiv. f. Gynäkologie*, 1906, Band lxxvii, Heft 3) from an examination of twenty-three dermoids concludes that in many cases an ordinary ovarian cystoma is invaded by dermoid tissue, the common picture presented being the filling of the lymphvessels and spaces with fat, so that the tumor appears to be almost like a lipoma.

Premature Menopause.—Darcanve-Movroux (Thèse de Paris; Abstract in *Zentralbl. f. Gynäkologie*, 1906, No. 31) emphasizes the fact that premature menopause must be distinguished from amenorrhœa due to chronic or acute diseases. In some instances the early disappearance of the menses may be referred to reflex nervous influences, or to superinvolution of the uterus, but more often to imperfect development. Delayed puberty, scanty and irregular menstruation and sterility, without impairment of the general health, point to insufficiency of the ovarian functions.

Rupture of Pyosalpinx.—Lejars (*Zentralbl. f. Gynäkologie*, 1906, No. 35) calls attention to the rare occurrence of intraperitoneal rupture of pus tubes and the difference between the gradual ulceration of the wall of an old pyosalpinx and the acute perforation of a recent abscess. He reports two cases of the latter, treated successfully by operation. In neither was there any history of traumatism, but it was evident from an examination of the specimens that the perforation was due to an ulcerative process, as in gangrenous appendicitis.

Sclerosis of the Uterine Bloodvessels.—Wittek (*Monatschrift f. Geb. u. Gyn.*, 1906, Band xxiii, Heft 6) describes the histological appearances presented in four uteri removed on account of intractable hemorrhage. It was noted that the continued use of ergot only increased the menorrhagia. This was explained by the condition of the intima of the arteries which had undergone connective-tissue hypertrophy. The result of uterine contractions was simply to compress the veins and to cause venous congestion, while the arteries were not affected on account of the rigidity of their walls. In one case Wittek believed that the arteriosclerosis was due to vasomotor disturbances, in the other to senile changes. This factor in the causation of menorrhagia is more common than is generally supposed, nor is the arteriosclerosis necessarily of inflammatory origin. Since frequent childbearing is a predisposing cause, the presence of arteriosclerosis should be inferred in the case of multiparæ in whom no other obvious reasons for menorrhagia can be discovered.

OPHTHALMOLOGY.

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AND

T. B. SCHNEIDEMAN, A.M., M.D.,
PROFESSOR OF DISEASES OF THE EYE IN THE PHILADELPHIA POLYCLINIC.

Permanency of the Results in the Operative Treatment of High Myopia.—Pause (*Ztschr. f. Augenhk.*, 1906, No. 11) publishes a continuation of Pflüger's monograph, "Suppression du cristallin transparent," Paris, 1899, in an attempt to determine the permanency of the results in the patients operated on by Pflüger up to 1894; twenty-four of forty-six patients could be found for examination. With the exception of one patient in whom central vision was lost from extensive choroiditis, the visual acuity was increased twice and fourfold and in one patient fivefold. Improvement in the function of the retina is to be regarded as the cause of such increase in the acuity. Central recent and old choroidal changes are not to be regarded as contraindications; on the contrary, the operation tends rather to conservation of central vision (in two out of three cases observed). The increase in the anteroposterior axis is not inhibited entirely but only checked. All the patients were well pleased with the result; many claimed that their sight was worse in poor light. Fukala's method of operating has not thus far been surpassed by any other.

A Diagnostic Point in Sarcoma of the Choroid.—Purtcher (*Centralb. f. prak. Augenhk.*, May, 1906) emphasizes the diagnostic value of a dark deposit on Descemet's membrane and the iris in sarcoma of the choroid; these deposits somewhat resemble an hyphema and are due to migration of sarcomatous elements from the tumor into the anterior segment of the eyeball, especially into the anterior chamber. Such "coffee-ground" collections in the angle of the anterior chamber evidently predispose to the increased tension which so often accompanies intraocular growths.

Protection of the Eyes against Ultra-violet Rays.—Dor (*Revue generale d'Ophtal.*, October, 1905) calls attention to the well-known fact that the ultra-violet rays have an injurious effect upon the cornea, lens, and retina, in general—like that of snow blindness and strokes of lightning. The best protection against these chemically active rays is yellow glass, such as is used by photographers. Such glass should be employed in the first lenses given after cataract extraction. Fortunately, there are also certain kinds of smoked glass which, without being very dark, are quite impervious to the ultra-violet rays, as is easily proved by photography. Dor proposes the term "achemical" for such lenses.

Stilling's Theory of Myopia.—(*Ztschr. f. Augenhk.*, 1906, No. 1.) In view of the prevalent misunderstanding as to his real views, Stilling again gives a comprehensive exposition of his hypothesis as to the production of myopia. Myopia occurs, in so far as prolonged near work is its cause, from too great development of the length of the eye due to pressure of the external muscles. It is not to be supposed that the pressure of the muscles directly affects the change in the shape of the globe, but rather that the tendons under tension offer an obstacle to growth in directions at right angles to their planes. Inasmuch as this obstacle is least active in an anteroposterior direction the eyeball in its growth naturally follows this line of least resistance. All the muscles are active in coercing growth under their pressure, but the oblique muscles are the controlling factor because their action is variable, while that of the other muscles is constant.

Filaria Loa.—Vail (*Amer. Jour. Ophthal.*, December, 1905) reports the case of a wife of a physician who experienced painful sensation with redness of the right eye during her residence in Africa, in 1899. A worm-like movement was observed beneath the conjunctiva, which disappeared after two hours at the external canthus; two days later the same phenomena were noticed in the other eye. Subsequently similar vermicular movements took place beneath the skin of the upper lids. Her husband succeeded by incision in removing some of these small worms, which, moreover, appeared at numerous other parts of the body and could be in part removed. The worms manifested a special partiality for the lids and tissues near the eye. Several of these worms were later removed (the condition having continued unchanged for years) by the reporter, and recognized as Filaria loa.

Results of Removal of the Clear Crystalline in High Myopia.—De Font-Reault (*Annal. d'Oculist*, 1906, vol. cxxxv) analyzes the results given by various statistics with particular reference to cases which have been followed over five years. Myopes of 20 D. generally become emmetropic. Visual acuity for distance is most frequently increased, but the opposite for near. Progression of increase of the anteroposterior axis is generally greatly diminished in the non-operated as well as in the operated eye, due no doubt to the abolition of binocular vision. The operation appears to exert little influence upon the choroiditis present. Detachment of the retina is rather more frequent. The operation has sometimes caused glaucoma at a more or less distant epoch. He concludes that the operation should only be done in eyes having a myopia above 20 D. in subjects requiring an improvement in their distant vision, and that it should only be performed upon a single eye.

PATHOLOGY AND BACTERIOLOGY.

UNDER THE CHARGE OF

WARFIELD T. LONGCOPE, M.D.,

DIRECTOR OF THE AYER CLINICAL LABORATORY, PENNSYLVANIA HOSPITAL,

ASSISTED BY

G. CANBY ROBINSON, M.D.,

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Studies on Spirillum Obermeieri and Related Organisms.—Novy and Knapp (*Jour. Infect. Dis.*, 1906, iii, 291) have made a very extensive study of an organism causing relapsing fever in man. They offer a number of convincing proofs that Spirillum obermeieri is a bacterium and not a protozoon. It is probably related quite closely biologically to Treponema pallidum or Spirocheta pallida, and is very similar to the organism causing tick fever of Africa and Bombay relapsing fever, which facts add great importance to the work. The blood at the beginning of one of the paroxysms seen in relapsing fever has very little germicidal power, ana the spirillum will live in it in vitro as long as forty days. The organism rapidly dies, however, in blood withdrawn toward the end of a paroxysm, and the authors think that the course of the recurring paroxysms depend on a rise and fall of the germicidal properties of the blood. Men, monkeys, white mice, and grats are subject to the infection, although paroxysms do not occur in the last-named animal. All attempts at cultivation of the organism have proved unsuccessful. Both active and passive immunity was produced, rats being especially used, and a passive immunity could be established by injections of hyperimmunized blood. This procedure caused the spirilla to disappear rapidly from the circulating blood. In the hyperimmunized blood, and in the blood of animals which were recovering or had recovered from the disease, there is a germicidal substance that is probably distinct from the immune bodies which also exist. Active and passive immunity last about one month. Hereditary immunity can also be obtained and is probably due to intrauterine infection. Hyperimmunized blood has a distinct preventive and curative action in rats, mice, and monkeys, thus establishing a solid basis for the hope of prevention and cure of relapsing fever in man. This immunized blood also has a strong agglutinative property in vitro and in vivo. Agglutination occurs to a slight extent in the blood at the time of crisis in a paroxysm. The agglutinating property of the serum may be used in the serum-diagnosis of the disease, and also as a means of identifying the organism.

Novy and Knapp believe that there is probably a group of relapsing fevers, and that the disease seen in Bombay and the Tick fever of Africa are caused by organisms distinct from Spirillum obermeieri. They are inclined to believe that relapsing fever may be conveyed by insects, and this is the first evidence that insects may serve as a means of transmission of a bacterial infection. Up to the present time the proof that a disease was transmitted by insects was considered as strong evidence against bacterial infection. The authors believe it is not improbable that the cause of yellow fever may be an organism belonging to this group.

The Relation between the Duration of the Disease and the Age of the Intestinal Lesions in Typhoid Fever.—It is usually stated and generally believed that in typhoid fever the formation of the intestinal ulcers progresses coincidentally with the course of the disease, so that roughly the various stages in the development of the intestinal lesions correspond to definite periods of the attack of fever. Thus, the infiltration of the lymphatic apparatus marks the second week, necrosis occurs in the third week, the slough takes place in the fourth week, and after the fifth week there is clean ulceration. WENNAGEL (*Deut. Arch. f. klin. Med.*, 1906, lxxxvii, 551) finds that this is true only in about 50 per cent. of ninety cases of typhoid fever which he has studied in Strassburg. The difficulty of finding out the day of onset is always to be taken into consideration, but Wennagel decided that the most accurate method was to count from the day on which the patient noticed his first symptoms, such as headache, malaise, etc. Accordingly, it was found that the stage of infiltration of Peyer's plaques occurred not only in the second, but in the third, fourth, and fifth weeks; and the stage of slough formation, sloughing, and clean ulceration from the second to the seventh week. Infiltration was found oftenest, however, in the second week (42 per cent. of the cases). Wennagel concludes that in the early stages of the disease advanced lesions, such as slough formation, do not occur. Before the third week there are seldom clean ulcers. Further than this it is not possible to determine from the anatomical findings at autopsy with any degree of accuracy the duration of the disease. Typhoid fever should be considered a general infection in which intestinal lesions may appear.

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THE
AMERICAN JOURNAL
OF THE MEDICAL SCIENCES.

FEBRUARY, 1907.

ORIGINAL ARTICLES.

THE SYMPTOMATOLOGY, DIAGNOSIS, AND SURGICAL TREATMENT OF CERVICAL RIBS.

By W. W. Keen, M.D.,
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I HAD never seen a case of recognized cervical rib until October 20, 1905. Within less than a month, personally or through Drs. Hearn, Patterson, and Taggart, of the surgical department of the Jefferson Medical College Hospital, I saw four other suspected cases, of which two proved not to be cervical ribs and two others were. Of the last two, only one needed operation (Patterson's). The history of the patient operated on by myself is as follows:

Mrs. M. A. R., aged sixty-three years, entered the Jefferson Hospital October 20, 1905, at the request of Dr. F. R. Brunner, of Eshback, Pa., who kindly furnished me with the following facts:

Her father died of some disease of the heart and her mother of typhoid fever. Nine brothers and sisters are living and well.

In childhood, she had measles, mumps, whooping-cough, chickenpox, and scarlet fever, and at the age of twenty-three years, typhoid fever. From all of these she made good recoveries. She has had several attacks of rheumatic fever. She was married at thirty, and has had one child. Her general health has been very good.

At the age of seventeen years she noticed a lump above the left clavicle. Although its presence did not cause her any trouble, she consulted a physician, who told her it was a "bone." She never had any trouble from it until last February, when, upon rising in the morning, she noticed that the first and second fingers of the left hand were the seat of sharp stinging pain, and that they were much paler than the others and cold to the touch. Brisk friction for a few moments would cause these symptoms to disappear. About a week later the thumb and the remaining fingers also became involved. A few days later she complained of a constant sharp stinging pain in the left hypothenar eminence. In the course of a few days this pain extended up the ulnar side of the forearm to the elbow, the radial side becoming involved soon afterward. Later, the pain extended up the arm to the shoulder. It was so severe as to cause her to lose considerable sleep. With the onset of the pain, the left hand became distinctly less red than the other. During last June the pain gradually disappeared. About the first of September, she again noticed a tingling sensation and shooting pain in the finger-tips of the left hand, and that the fingers were red and slightly swollen. About two weeks later the soft parts at the tips of the fingers, especially under and about the nail, became gangrenous in the following order: index, ring, thumb, middle, and little finger. She stated that the pain was always less severe while she used the hand freely in her work.

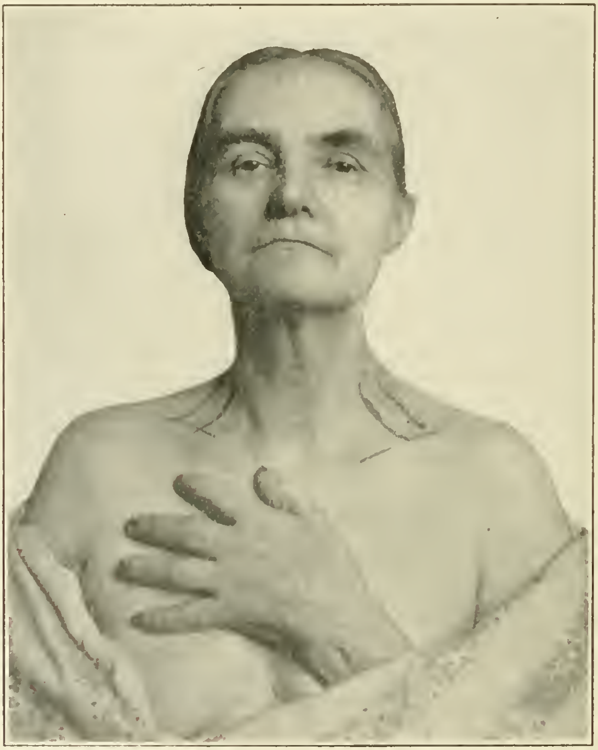

FIG. 1.—Photograph of Keen's case of cervical ribs. The obliquely horizontal lines correspond to the clavicle. The double obliquely vertical lines correspond to the subclavian arteries. The gangrene of the finger-tips shows imperfectly.

Physical Examination on Admission. Weight 119 pounds. There is no arcus senilis present. There is a soft systolic cardiac murmur heard best at the apex. The lungs are apparently normal. There is no pulse whatever in the axillary, brachial, radial, or ulnar arteries on the left side. The left subclavian can be felt above the left clavicle running downward and outward to the clavicle for a distance of 6 cm. (Fig. 1). Its width as ascertained by putting one finger upon each side of the vessel, and including, of course, two layers of skin and subcutaneous tissue, is 18 mm. It pulsates very strongly to the touch. Though there is no thrill, a stethoscope applied over the artery reveals a local bruit which is not propagated. Behind the artery is a rounded swelling above the left clavicle a little internal to the junction of its middle and outer thirds. On deep palpation this swelling is found to be part of a hard bony mass which can be traced backward to the spinal column. As it curves forward it seems to pass behind the clavicle. The artery passes in front of (that is, over) this bony prominence, which was diagnosticated as a cervical rib.

The left hand is distinctly cold and very much paler than the right. The nail of the middle finger is entirely loose and there is an ulcerated surface corresponding to the site of the nail, passing through the tip of the finger midway between the dorsal and the plantar surfaces. The other four nails are discolored and the tips of the fingers are somewhat ulcerated, the worst being the ring finger. This ulceration is the result of the sloughing off of the slight gangrenous patch from the subungual space (Fig. 1).

A slight bruit can be heard over the right subclavian, but it is much less distinct than that over the left. There is no thrill on either side.

Behind the right subclavian artery also a cervical rib is present and can be traced back to the spine. Both of the cervical ribs are immobile. A skiagraph (Fig 2) shows bilateral cervical ribs, the left being the larger rib.

The right subclavian artery also runs much higher than is usual and can be traced upward from about the junction of the middle and outer thirds of the right clavicle for a distance of 3.5 cm. Its width, measured as already estimated on the opposite side, is 15 mm. These measurements for the diameter of the vessels naturally should not be taken too exactly. The pulse on the right side is normal. The muscles of the left shoulder and the entire arm are considerably wasted. This is due in some measure to non-use of the hand, for all of the joints of the fingers, from having been kept of late quiescent and part of the time poulticed, have become partially ankylosed.

The right arm at the middle of the biceps measures 24.5 cm.; the left arm, 24 cm.; the right forearm, 8 cm. below the external condyle 20 cm.; and the left forearm, 8 cm. below the external condyle 18 cm.

Neither inspiration nor elevation of the right arm has any effect upon the pulse. The urine is normal except for a rather low percentage of urea.

Dr. Alfred Gordon kindly examined her, and confirmed the above findings and also found marked tenderness and hyperalgesia in the hand. Sensation in the arm and forearm is very slightly diminished. The temperature sense is normal.

Left side

Right side

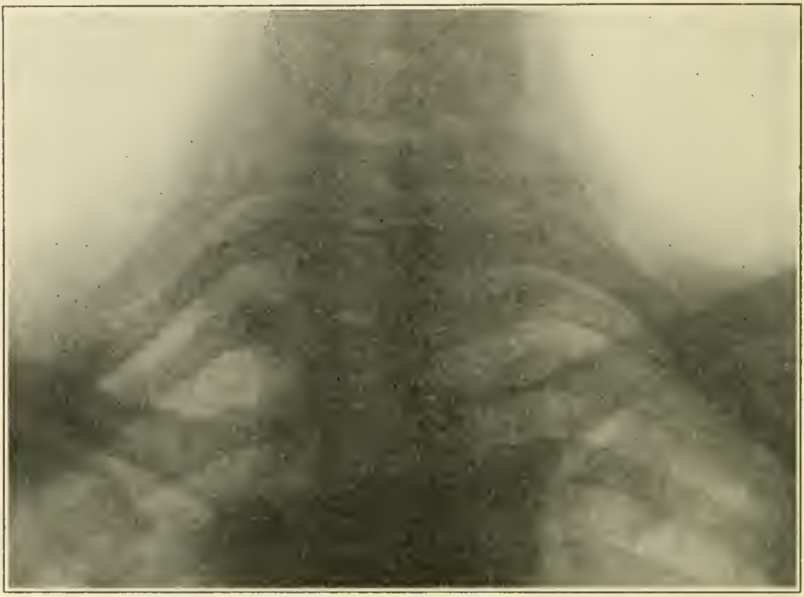

Fig. 2.—Skiagraph of Keen's case, showing bilateral cervical ribs, the left being the larger and higher and the only one causing symptoms.

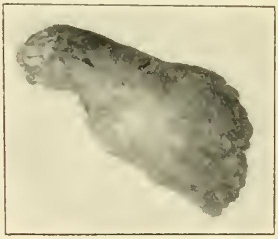

Fig. 3.—The piece of rib removed in Keen's case (natural size). Note the knob at about its middle. This formed the tumor felt behind the subclavian artery.

Operation November 29, 1905. An incision just above the left clavicle and parallel with it, a second vertical incision directly over the tumor, and dissection of the internal and external flaps, gave ready access to the tumor. This was found to consist of the subclavian artery and immediately back of it a cervical rib—attached to the seventh cervical vertebra and immovable. The attachment to the first rib just behind the middle of the clavicle was broad, and relatively thin, partly fibrous, but chiefly bony. The middle of the rib consisted of a large knob of bone (Fig. 3). The brachial plexus as well as the subclavian artery ran over the cervical rib. The cords of the plexus instead of being cylindrical were markedly flattened. By displacing the artery first inward, then outward, I was able to expose thoroughly the anterior end of the bone, to which part the scalenus anticus was attached, and to detach this muscle from the bone by scissors. Then displacing the artery internally and the nerves externally, I was able to uncover the posterior end and separate the surrounding tissues from it. With a pair of bone forceps I divided the bone posteriorly and anteriorly, removing the periosteum with it. The fibrous part of the anterior end was divided by scissors, the parts underneath being guarded by the handle of an instrument. I was particularly careful at this point, fearing every moment that I should tear the dome of the pleura. At no time, however, did I see the pleura nor was it injured, nor was the subclavian vein seen at any time. With a pair of rongeur forceps I then smoothed off the two ends of the bone. The artery where it emerged from behind the scalenus anticus seemed to be smaller in diameter than usual. It changed its course downward and outward abruptly by a sharp bend (Fig. 4). Immediately beyond the external border of the muscle it was dilated to nearly twice the

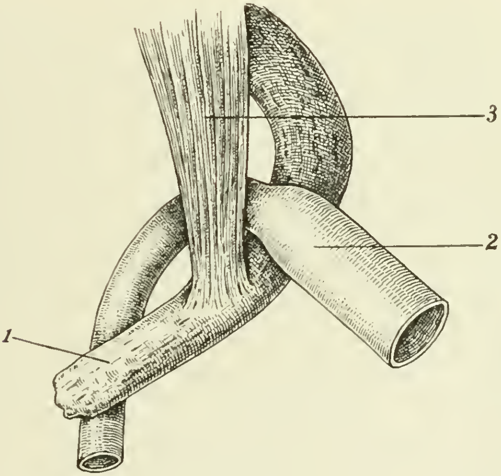

Fig. 4.—Keen's case. Sketch showing the marked increase in size of the subclavian artery after escaping from behind the scalenus anticus. 1. Cervical rib. 2. Subclavian artery. 3. Scalenus anticus.

width of the vessel as it emerged from behind the scalenus anticus. Down to the point where it disappeared behind the clavicle it retained this enlarged diameter. Beyond that point it could not be followed.

A small cigarette drain was placed in the wound, which, after douching with salt solution, was closed by ordinary sutures. The patient was placed in bed in admirable condition.

In the afternoon of the operation her temperature rose to 100.6°. It was down to normal on the second day, and so continued until she went home. The cigarette drain was removed at the end of twenty-four hours, and the wound healed without note. I desired later to etherize her and break up the adhesions in the finger-joints so as to give her a more useful hand, but she was unwilling that this should be done.

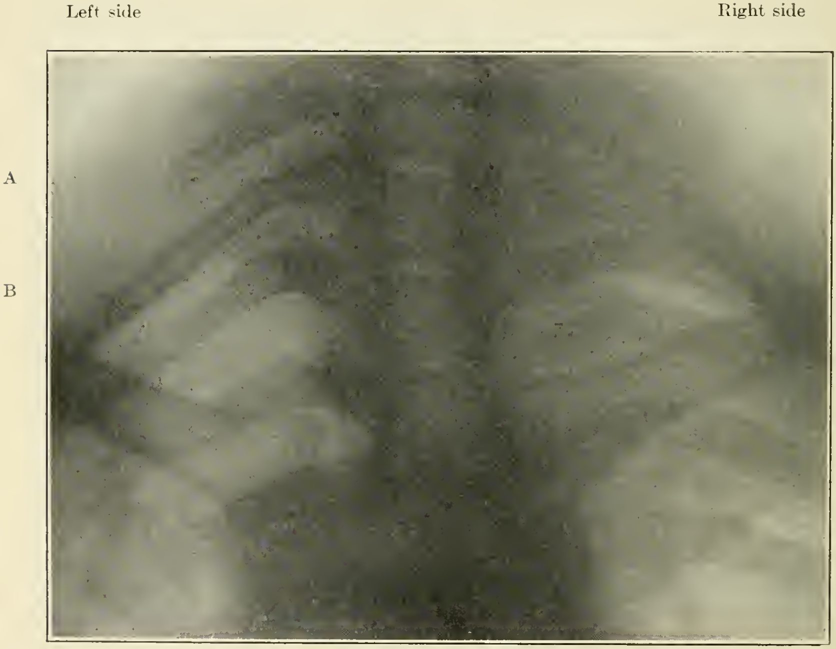

Fig. 5.—Keen's case. Skiagraph showing the gap in the cervical rib left by the resection Opposite A and B are seen the stumps of the two ends.

Immediately after the operation all pain disappeared from the left arm, and remained absent up to the time when she left the hospital, twelve days after operation. The bruit still persisted in the left subclavian, but there was no thrill. Pulsation was less evident than before the operation. The pulse did not return in any of the arteries below the subclavian.

Within twenty-four hours after operation the left hand assumed a much better color, and in the course of two or three days its temperature was almost the same as that of the right hand. Meantime the ulcerated spots on the fingers dried up and crusted over, so that she was no longer obliged to protect them even with a dressing.

A new skiagraph (Fig. 5) shows where the resection left a gap in the left cervical rib.

October 20, 1906. Eleven months after operation, Dr. Brunner reports that she does not suffer at all except from slight pain in the forearm when she works much. Her sleep is excellent. The subclavian is normal. There is very slight pulsation (?) in the axillary and ulnar arteries, none in the radial. The nail is absent only on the middle finger; the fingers are not painful. She cannot quite close them, but she uses her hand for all kinds of work, and it feels the same and is as strong as the other.

A cervical rib is a congenital vice of development. Unlike most other such defects, it usually exists alone. But it may be associated with other congenital defects. Thus, Ballantyne[1] mentions a case, a woman, aged twenty-two years, in which there was an imperforate vagina with distention of the uterus. Poland describes a case associated with club-foot. Borchardt, Oppenheim, Marburg, and Schönebeck mention cases associated with syringomyelia. Levi describes one with the symptoms of multiple scleroses; Spiller and Gittings published a case of progressive muscular atrophy of cervicobulbar type similarly associated. Most of these were defects of development of the central nervous system. I have not found any other defects either in the skeleton or the soft parts associated with cervical rib. Though the defect is congenital the symptoms, as I shall point out later, seldom develop early in life.

Most cases of supernumerary cervical rib are attended with very slight or more frequently with no symptoms whatever. In fact, the existence of the anomaly is often entirely unknown until discovered either by reason of some illness requiring examination of the neck, at a postmortem, or in the dissecting-room. It is often assumed that the specimens found in the dissecting-room have caused no symptoms. It is, however, by no means a certainty that they were without symptoms, for the clinical history of the cadavers is rarely known.

Although mentioned by Galen (see Hunauld) and other early authors, our real knowledge in reference to the existence of cervical ribs dates from the paper by Hunauld, published in 1742 (Fig. 6). In 1849, Gruber published a paper on "Cervical Ribs," and twenty years later, in a second paper, increased the number of known cases to seventy-six. He quotes the first two cases (Willshire and Huntemüller) in which the anomaly was recognized in the living. Three other cases recognized during life were added by Struthers, in 1875. In 1894, Pilling increased the number of reported cases to 139, of which nine had been recognized during life.

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[1] All the references are in alphabetical order at the end of the paper.

Since that time a large number of cases have been reported. Most of them are interesting only from the anatomical and embryological point of view.[2] These I do not propose to consider, but shall limit this paper chiefly to those which have been treated by operation, incidentally considering a few of the other cases which are of interest from the surgical point of view, either by reason of their symptoms, physical signs, or treatment. The appended list contains all the cases subjected to operation which I have been able to find, some of them from histories kindly furnished by the authors, to whom my thanks are due.

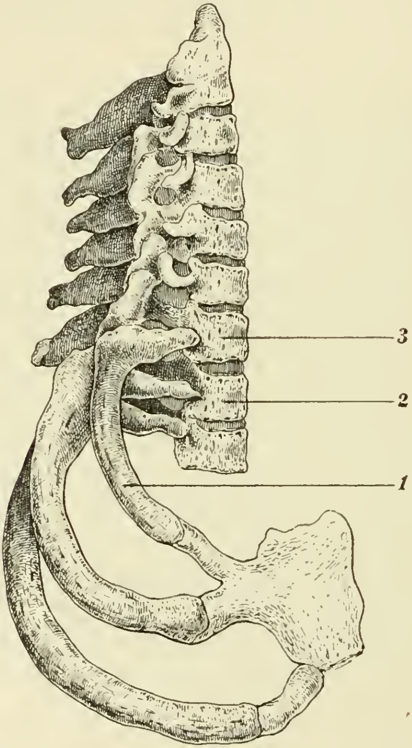

Fig. 76.—Showing a complete cervical rib. Cervical rib. 2. First dorsal vertebra. 3. Seventh cervical vertebra. (Hunauld.)

One of the chief reasons for the large increase in the number of cases reported in recent years is undoubtedly the discovery of the x-rays. In suspicious cases, examination by this method enables us to reach a decision whether or not such an anomaly exists. As a result of this positive method of diagnosis many cases which would otherwise have escaped observation have been definitely proved to be genuine.

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[2] Readers desiring to look these up should consult the bibliographies of Krausse (the fullest to date), Schönebeck, Eisendrath, and Riesman.

Age. The age at which cervical ribs have been discovered varies from the sixth month of intrauterine life (Rosenmüller, Pilling) to old age. The age when the cervical rib produces symptoms, however, is far different. (*Vide infra.*) Symptoms rarely manifest themselves until adolescence. In other cases they do not arise until much later, though the patient has borne the rib since birth. The reason for this late onset of symptoms will be discussed later.

Sex. Why there should be such a disparity between the two sexes it is impossible to say, but it is at least curious to note that it is much more frequently met with in the female than in the male sex.[3] Among the 42 patients operated on, which I have collected, there were 31 females and only 11 males.

Side. Naturally one would expect the anomaly to be nearly, if not quite, equally frequent upon the two sides. As a matter of fact, we find that up to the time when Pilling collected his 139 cases (1894) it was believed to be bilateral in only about two-thirds of the cases. In all probability this does not represent the real percentage, because the smaller rib from its non-production of symptoms and difficulty of discovery has often been overlooked. The *x*-rays have demonstrated the far greater frequency of their bilateral existence. Thus, Schönebeck, in 1905, states that of eighteen skiagraphs, fourteen showed bilateral and only four a unilateral anomaly. In none of the fourteen was the rib equally developed upon both sides. Usually only the larger rib produces any symptoms. In fact, so far as I know, there are but two cases in which symptoms were developed on both sides (Rutkowski and Schönebeck).

In two cases there were two ribs on one side. Struthers records a case of rudimentary bilateral cervical ribs on both the sixth and seventh cervical vertebræ in the cadaver of a boy, aged four years, and Ehrich records another remarkable and, indeed, unique case, operated on by Karg, in which there were two cervical ribs attached to the sixth and seventh vertebræ. The subclavian artery passed *between* the two ribs in so narrow a space that full inspiration entirely obliterated the radial pulse. Stifler, in 1896, reported a case in which he believed the rib arose from the sixth cervical, but in the absence both of operation and a skiagraph, it is permissible to doubt. Beck believes that in his case there was a rudimentary rib arising from the sixth as well as from the seventh vertebra. In Hauswirth's and Williams' cases, the rib was erroneously reported as arising above the seventh vertebra.[4]

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[3] Drs. Thomas and Cushing (Johns Hopkins Hosp. Bull., November, 1903, xvi) call attention to an interesting passage in Sir Thomas Browne's Pseudodoxia Epidemica or Commentaries on Vulgar Errors, London, 1650, 292, on the supposed difference between the female and the male skeleton as to supernumerary ribs, which caused a very heated discussion based on the Biblical story of the creation of Eve. However, this, like Eve, is a "side issue."

With these few exceptions, the rib has always arisen from the seventh cervical vertebra.

Were this anomaly entirely an anatomical one, it would possess but little interest to the surgeon other than in operations on the subclavian artery or the brachial plexus. As in the cases of cervical rib, the subclavian artery lies much higher in the neck than usual, it lessens the difficulties of reaching the artery, but might mislead the surgeon were he not aware of the anomaly. Stanley Boyd mentions one case in which it was an advantage, as compression of the artery for aneurysm was greatly facilitated by the greater accessibility of the artery. But the cases in which symptoms arise may prove exceedingly serious and become of importance to the surgeon as well as to the neurologist, internist, and pathologist. The pain and disturbances of sensation which occur, the serious wasting of the muscles of the arm, the diminished if not total loss of usefulness of the arm, the partial or total obliteration of the pulse, the occasional gangrene of the fingers, and the occasional occurrence of aneurysm all make the lesion a matter of great surgical importance.

ANATOMY. A cervical rib is only an abnormal development of a normal portion of the vertebra. We are apt to forget that in the cervical, the lumbar, and even the sacral region there exists a representation of that which in the dorsal region is fully developed into a normal rib.

In the accompanying diagrams (Fig. 7), from Morris' *Anatomy*, one can see that while the "costal process" is fully developed into a true rib only in the thoracic region, potential ribs exist in all the other three regions of the spine in a rudimentary form.

It is interesting to observe that the most frequent site for the extra rib is from the lowest or seventh cervical vertebra, that is, the point of transition from the last cervical to the first dorsal vertebra, very rarely from the sixth, and only in one case (Völcker) above that. Occasionally also supernumerary ribs are developed in the lumbar region adjacent to the last dorsal rib.

The development of the cervical rib may vary simply from a slight

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[4] I have not included in my table of cases of cervical rib which have been operated on the case of Czerny and reported by Völcker, as the lipoma was the chief lesion and the rib was rudimentary and incidental. This very remarkable and unique case was one of fatty tumor of the neck, in a girl of fourteen. The tumor weighed a kilogram, was chiefly a deep lipoma, with areas of calcification and ossification. The pedicle of the tumor was a rudimentary rib connected with the transverse process of the fifth cervical vertebra. Völcker calls attention to the fact that lipomas frequently take their origin from congenital vices of development. He believes that in this case in all probability the tumor began in intrauterine life. This is the only case reported in the literature of a deep lipoma of the neck connected with the vertebral column and the only case of a rib arising above the sixth vertebra. There was no additional rib from the sixth and seventh vertebræ.

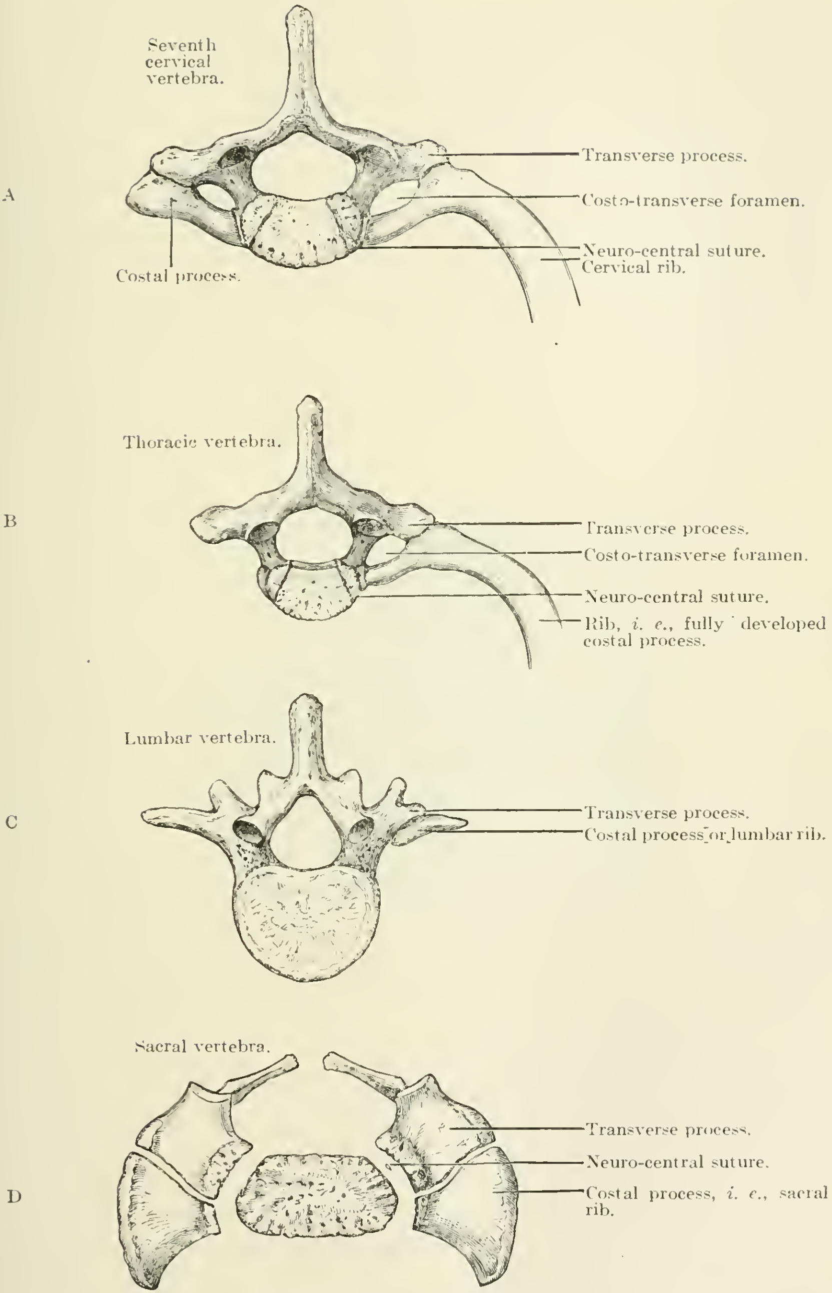

FIG. 7.—Note in A, on the right side of the seventh cervical vertebra, the costal process which on the left side is represented as a cervical rib. This costal process, or potential rib, exists in all the cervical vertebræ. In B, a thoracic vertebra, the fully developed costal process is a normal rib. In C a lumbar, and D a sacral vertebra, the costal process, *i. e.*, rib, exists in an undeveloped form. (Morris' Anatomy.)

exaggeration of the costal process of the seventh cervical all the way to a complete cervical rib attached to the sternum. Imperfect ribs are much more common than completely formed ribs. The best classification is that of Gruber into four degrees: First degree, a very slight increase of the costal process not reaching beyond the true transverse process. Second, a rib protruding beyond the transverse process to a moderate extent and ending either free in the tissues or attached in some way to the first thoracic rib. Third, a still further degree of development in which the cervical rib extends for a considerable distance toward or even to the cartilage of the first thoracic rib, possesses a complete body, and is united directly or by means of a ligament with the cartilage of the first rib. Fourth, a complete cervical rib uniting at the anterior extremity with the cartilage of the first rib through which it reaches the manubrium (Figs. 6 and 14.)

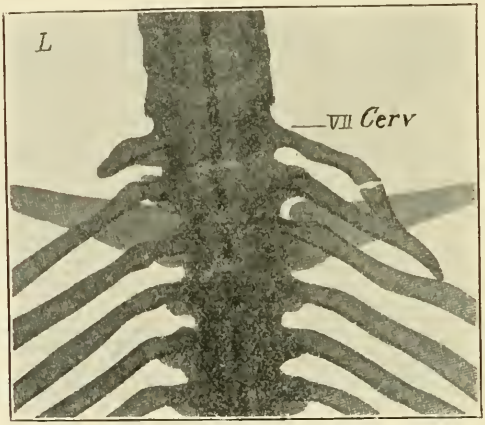

FIG. 8.—Note the very rudimentary rib on the left side; the larger one on the right side; the exostosis from the first dorsal rib with which the cervical rib articulates by a joint (Meyerowitz's Case 2.)

Dr. Lewellys F. Barker (an interested observer), in a private letter, states: "When I was in Europe, in 1904, I visited several anatomical museums and looked over the collections of cervical ribs. The best collection I saw was in Prague. The ribs showed the most remarkable diversity. Sometimes a cervical rib articulates behind with the body of the vertebra, sometimes with the intervertebral disk; in front it sometimes articulates with the first rib, sometimes with the sternum. Not infrequently there is extensive bony union between the cervical and the first rib. The length varies greatly. I saw very short nodules and all gradations between these and ribs running all the way to the sternum."

This is the fullest statement I have seen as to the variation in the vertebral attachment of the rib, except the important paper by

Drehmann. (*Vide infra* under Scoliosis.) It is to be noted that the vertebral attachment is sometimes by means of a complete joint, so that the cervical rib, like the dorsal ribs, is movable. In other cases there is no articulation; in this case the rib is immovable. Of course, if the anterior attachment of the rib is by a bony connection with the first rib, this also will render the tumor in the neck immovable. In several cases a joint has been noticed some distance from the first rib, in which case the specimen might be described as a cervical rib articulating with a longer or shorter exostosis from the first rib (Fig. 8). Not uncommonly this projection from the first rib, which, so to speak, rises up to a greater or less extent to meet the rib, is apt to be thickened at the point of junction, and produces a tumor consisting of two portions nearly at right angles to each other (Figs. 9 and 10; *cf.* Weissenstein, Ehrich (Madelung), de Quervain, Ranzi, Adams, Aron, Tilmann, and Borchardt). Commonly in the living subject the rib and the artery can only be traced to the level of the clavicle. Behind this they disappear and the anterior attachment of the rib can only be discovered by operation and sometimes not even then.

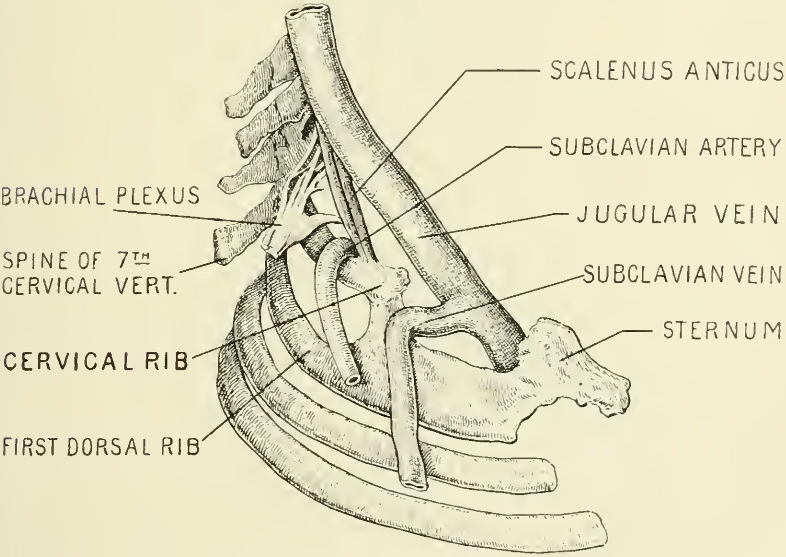

FIG. 9.—Rib and soft parts in the case of Weissenstein. Note the angle of the cervical rib, the sharp bend of the subclavian artery behind the scalenus anticus, and the position of the vein and the plexus.

When cervical ribs are developed on both sides not only is one almost always more highly developed than the other, but one is apt to be at a higher level than the other (Fig. 11). The symptoms then arise on the side of the larger and the more highly placed rib.

The most important relations of a cervical rib are to the artery, the vein, the brachial plexus, the muscles, and the pleura.

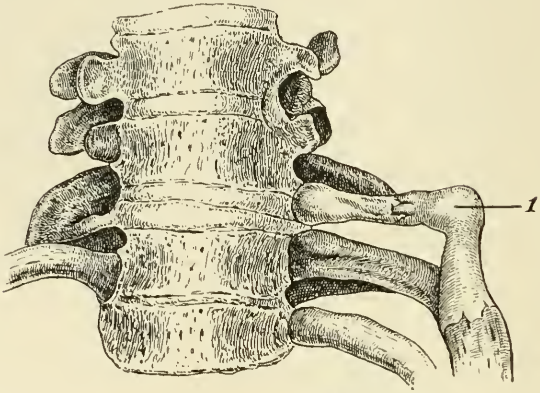

Fig. 10.—Sketch of the rib in the case of Ehrich-Madelung, showing the angular shape of the rib, its articulations, and the knob (1) at the angle.

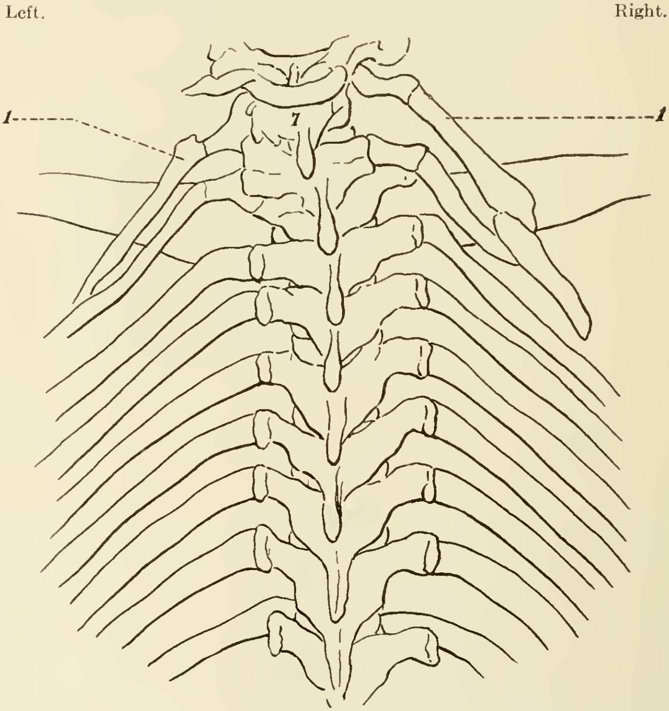

Fig. 11.—1, 1, cervical ribs. 7. Seventh cervical vertebra. Note how much higher the right cervical rib is than the left. Symptoms existed only on the right side. (Gordon, Brit. Med. Jour., June 8, 1901, 1395.)

The Artery. If the cervical rib is short or of only moderate development the plexus and the vessels do not pass over the rib, but in front of its anterior end. The rule promulgated by Halbertsma (that when the length of the rib is under 5.1 cm. the artery passes in front of and not over it, but when the length is 5.6 cm. or more the artery passes over it) does not literally hold good, but within certain limits it gives a very fair idea of what generally occurs. Gruber, Aron, and Brodier have reported cases in which the rib was 5.6, 5.75, and even 7 cm. long, yet the artery did not pass over the rib. The artery never runs under the cervical rib. When the rib is of considerable length so that the artery and nerves pass over it, the chest cavity is lengthened by the addition of this rib; hence, the artery is no longer than normal, rises much higher in the neck than normal, and after it crosses the extra rib, passes downward at a much more acute angle than usual. Ordinarily in order to compress the subclavian artery it is necessary to insert the thumb or finger well behind and beneath the clavicle, but as will be observed by looking at Fig. 1, in my own case, in which I outlined the length, diameter, and direction of the subclavian, it will be seen how far above the clavicle the artery passes and how oblique is its course. This photograph emphasizes the accessibility of the artery for compression in cases of aneurysm.

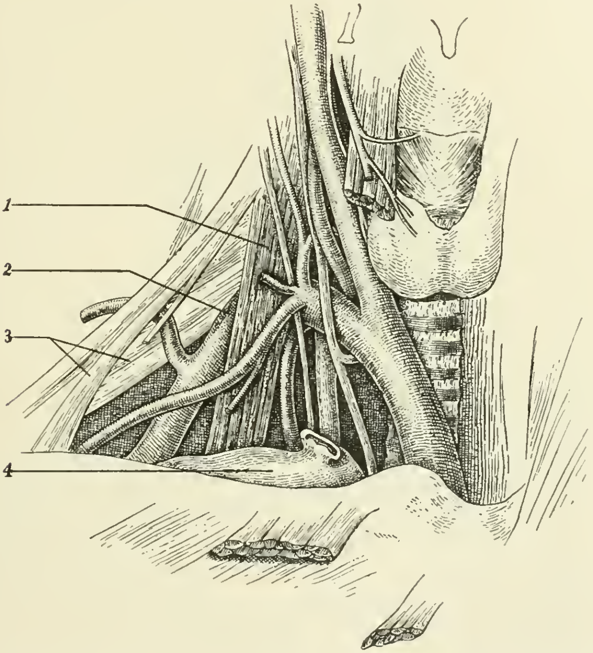

Fig. 12.—Showing that the subclavian artery may rise abnormally high in the neck, even when there is no cervical rib. Note also the position of the vein (Quain). 1. Scalenus anticus. 2. Subclavian artery. 3. Brachial plexus. 4. Subclavian vein.

But it must be remembered that even when there is no cervical rib the subclavian artery may pass high up in the neck. Thus, in the cases of "C. L." and "C. F. M." (*vide infra*) the artery rose 3.5 cm. above the clavicle. Quain (*Anatomy of the Arteries*, Plate XX, Fig. 3) also gives a case (Fig. 12) in which the same high position of the artery was shown by actual dissection, yet there was no cervical rib present.

The Vein. As a rule this is at a considerable distance in front of and below the artery. This is well shown in Fig. 12, and also in Fig. 13, from one of Broadbent's cases. This position of the vein, and, as insisted upon by Murphy, its not being compressed between the cervical rib and the scalenus anticus, since it lies in front of this muscle, account for the absence of œdema in the arm in nearly all cases.

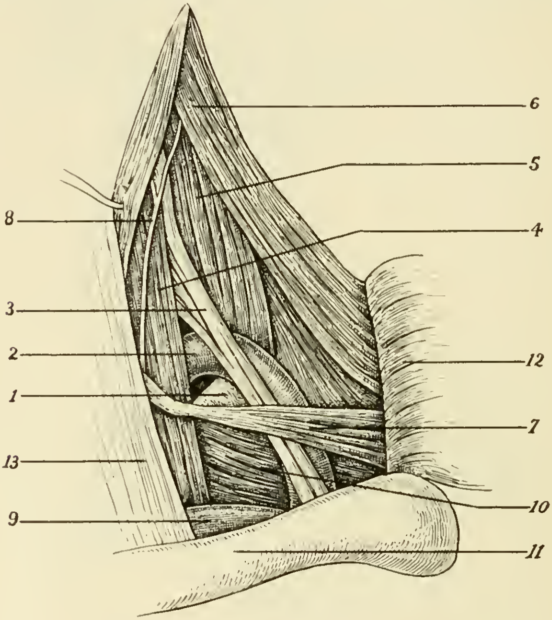

FIG. 13.—Showing especially the relation of the vein. 1. Cervical rib. 2. Subclavian artery. 3. Brachial plexus. 4. Scalenus anticus. 5. Scalenus medius. 6. Levator anguli scapuli. 7. Omohyoid. 8. Phrenic nerve. 9. Subclavian vein. (Broadbent, Brit. Med. Jour., May 5, 1906.)

The Brachial Plexus. As the brachial plexus lies above and to the outside of the subclavian artery, whenever the artery passes over the rib, the nerves must also pass over the rib.[5]

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[5] In Murphy's papers his artist has made two errors in the figures. An aneurysm is represented in one figure on the right side, and in another on the left side. In a personal letter Dr. Murphy states that the enlarged artery was on the left side, but that there was no aneurysm. The other error is more important. In Fig. 5 of the earlier paper and Fig. 3 of the later, the artist has represented the cross-section of the rib as a vertical instead of a horizontal oval, the artery to its outside instead of above it and the plexus below the artery and on the inside of the scalenus anticus, whereas the plexus really lies to the outside of and above the artery and wholly external to the scalenus muscle.

It is very important to determine the relation of these structures to the rib during operation, so that without injuring them we may gain easy access to the bone and remove it. Commonly the plexus is best displaced upward and outward, the artery downward and inward. Rarely will the vein require any attention, as it is not in the way. Indeed, often it is not even seen.

The Muscles. When the rib is developed to any considerable extent there is present an intercostal muscle, usually the external only. The scalenus anticus is often inserted into the anterior extremity of the cervical rib. *This is a clear evidence of the congenital origin of the anomaly.* The medius and posticus may either escape insertion into the cervical rib or be inserted into this rib alone, or they may be inserted into the cervical and the first and second dorsal ribs. Occasionally (Fig. 14) the artery and nerve penetrate through the scalenus medius, dividing it into separate fasciculi. The importance of the muscular relation of the artery will be seen later.

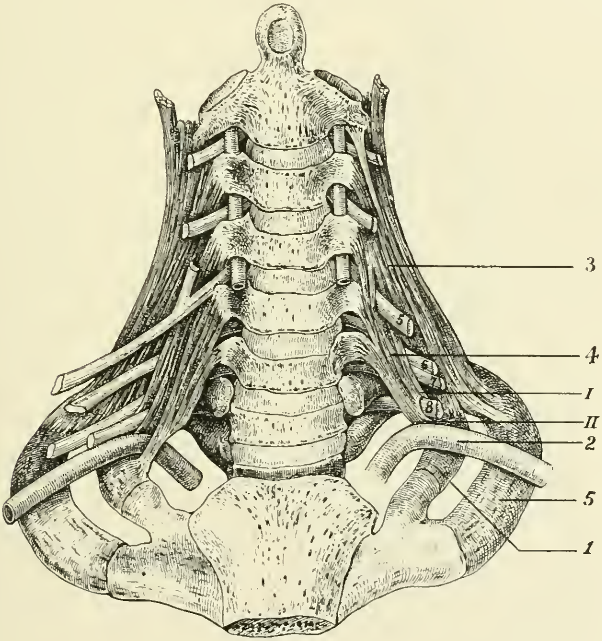


Fig. 14.—Showing division of the scalenus anticus into bundles by the artery and the nerves on both sides (Pilling). 1. Cervical rib. 2. Subclavian artery, which here shows in *front* of the scalenus anticus instead of behind it. 3. Scalenus medius. 4. Scalenus anticus, divided by the artery and the nerves into bundles. 5. First dorsal rib. I and II. Two bundles of the scalenus anticus on the left side; on the right side it is divided into three bundles. The artery could be compressed by the scalenus anticus on the right side, but not on the left. The roots of the brachial plexus are numbered 5, 6, 7, and 8.

The Pleura. The pleura rises much higher in the neck in cases of cervical rib than in the normal neck (Fig. 15). Its arched dome is often so closely adherent to the rib that it is very difficult and sometimes impossible to separate them without tearing the pleura. In other cases, as in my own, it is so easily detached that it may not even be seen. Its proximity must always be borne in mind and great caution exercised when dissecting the rib on its under surface.

SYMPTOMS. The symptoms may be grouped under four headings: (I) Local symptoms in the neck; (II) the nervous symptoms; (III) the vascular symptoms; and (IV) the muscular symptoms.

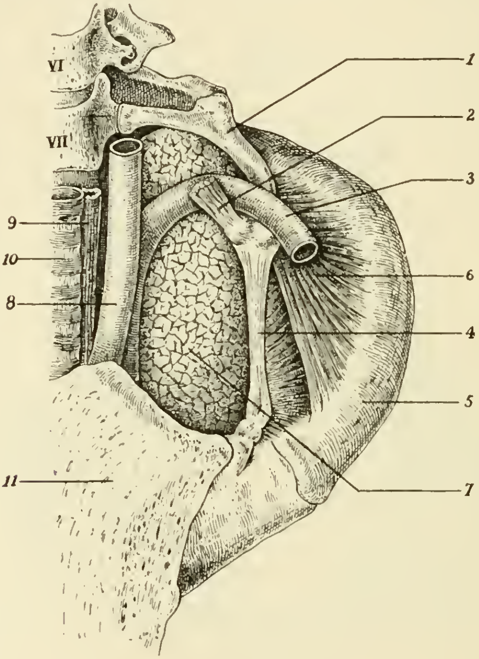


FIG. 15.—Showing a rudimentary rib (1) with its knobbed end, to which is attached the scalenus anticus (2). The rib is prolonged to the sternum by a ligamentous cord (4). Note *the relation of the pleura and lung* (7) *to the cervical rib.* 3. Subclavian artery. 5. First dorsal rib. 6. Intercostal muscle between the cervical and the first dorsal rib. 8. Left carotid artery. 9. Œsophagus. 10. Trachea. 11. Sternum. (Luschka.)

I. *Local Symptoms.* There is usually a tumor in the neck. This may be either palpable, or visible, and frequently is perceptible to touch and sight. The site of the tumor is usually about one to two fingers' breadth above the middle of the clavicle. It is usually more or less rounded, hard, more frequently immovable, but sometimes, when there is an articulation at each end, slightly movable. It can be traced back toward or even to the seventh cervical vertebra. In addition to this, above the tumor, and, therefore, relatively high up in the neck and running obliquely from above downward and outward, there is a visible and palpable pulsation. A high and oblique pulsating vessel, if observed, should always arouse the suspicion of a cervical rib. The cause of this pulsation is the displaced subclavian artery. There may be a bruit, which, however, is not often propagated; rarely will there be a thrill. The pulsation is sometimes expansile, but more commonly it is not. Just above and to the outside of this pulsating vessel there is tenderness, and in very thin patients the seat of this tenderness, namely, the brachial plexus, is palpable. On the other hand, palpation is, in my opinion, a very misleading, and by no means reliable, method of examination. Dr. Patrick, of Chicago, in communicating to me the history of his first case (the first one, I believe, diagnosticated and operated on in Chicago) has also made the same observation. The diagnosis reached by palpation should always be confirmed, or, as is sometimes the case, refuted by a good skiagraph. If the rib is very small, it may easily escape attention even on a careful physical examination.

As illustrations of the impossibility of absolutely determining by palpation the existence of a cervical rib, I mention the following cases seen by myself:

Supposed Cervical Rib, there being no Pulse up to and including the Axillary Artery on both sides and later none in the Subclavian. A skiagraph showed no such Rib.

C. F. L., aged twenty-three years, was first seen December 9, 1905, with Dr. T. D. Taggart.

Dr. Taggart recently had percussed her chest and neck and then accidentally discovered, as he believed, that she had bilateral cervical ribs. Up to that time she had no knowledge that there was any trouble in her neck. She has had absolutely no symptoms either of pain or discomfort.

On examination I found what I too thought was a cervical rib on both sides, which could be traced back to the spinal column. The subclavian artery on the right side was 3.5 cm. and on the left 3 cm. above the clavicle. It pulsated normally. Below the subclavian, in the axilla, arm, and forearm, no pulse whatever could be discovered on either side. The resonance of the lung in the neck extended up to the level of the cervical rib.

January 9, 1906. Examination of an *x*-ray picture shows no cervical rib on either side (Fig. 16). Re-examination of the patient showed a hard supraclavicular tumor on the left side with a smaller similar one upon the right. This probably is the first dorsal rib more highly placed than usual. I could not now detect, on either side, pulsation either in the subclavian itself or in any artery below that in either arm, yet the circulation was not affected, the skin was not blue, the white spots caused by pressure disappeared as quickly as usual, and in every way the arm seemed to be normal.

October 26, 1906. The same conditions as noticed in January still exist.

Partially Developed First Dorsal Rib Mistaken for a Cervical Rib. A Bruit, but no Perceptible Thrill in the Subclavian Artery.

C. F. M., aged twenty-one years, telegraph operator, was first seen January 30, 1906. About eight years ago he noticed an enlargement on the left side of the base of the neck. No other symptoms appeared until four months ago. Since then he has had a sensation of numbness in the left arm. It sometimes extends downward and involves the flexor surface of the forearm. During the past four months he thinks that the mass he observed has increased in size.

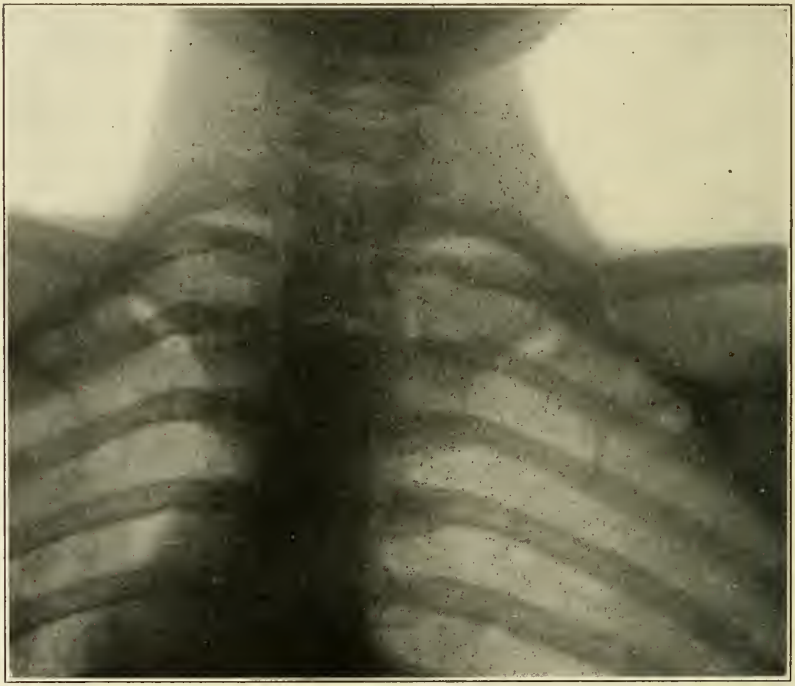


Fig. 16.—Case of C. F. L. Supposed cervical rib. Skiagraph shows none, yet there is a hard tumor in the neck and no pulse in either arm from and including the subclavian. (Keen.)

On examination the left side shows a fulness and the subclavian artery can be seen pulsating directly under the skin. Careful palpation shows that the subclavian artery appears in the neck at a point 3.5 cm. above the upper border of the clavicle and runs downward and outward, disappearing behind the clavicle. Palpation also shows behind and below the artery a hard, immovable mass which I believed to be a cervical rib. It could be traced backward to the spinal column and forward to the clavicle, behind which it was lost. At this time I did not make a careful examination of the first rib at the normal point for its attachment to the sternum. No thrill could be observed in the artery, but there was a distinct bruit observed by the stethoscope. Evidently the artery passed over the first rib. Its diameter also seemed to be increased. The pulsation in the two arms was normal. The right side of the neck showed no such prominent hard mass, no pulsation was visible, no bruit, and the subclavian artery ran behind the clavicle at its usual level.

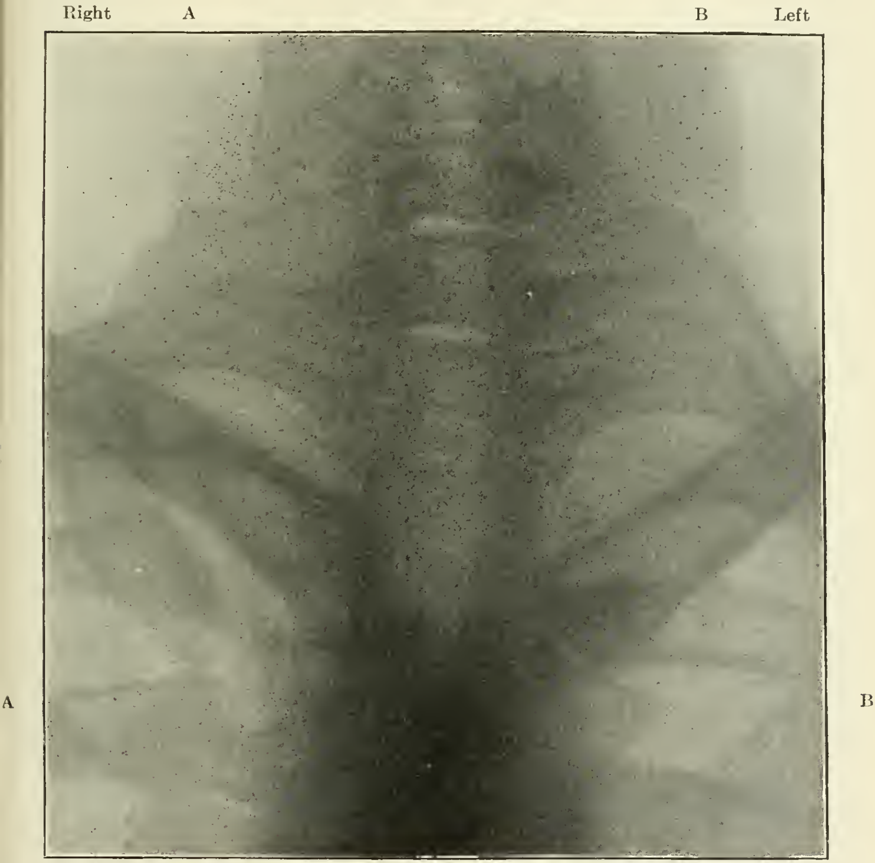


Fig. 17.—Partially developed first dorsal rib on the left side mistaken for a cervical rib. (See text.) (Keen.)

Dr. J. C. Da Costa, Jr.. kindly examined his blood pressure and found a systolic blood pressure in the normal position of the left arm, of 132 mm. With the patient sitting upright and the arm raised above his head it was 125 mm.

I directed that a skiagraph should be taken. On examination a very unusual anomaly of the first rib was observed.

The supposition that the patient had a cervical rib was proved by the skiagraph to be incorrect. On the right side (Fig. 17) the first rib is very clearly seen and at the point where the two lines A A would cross, the cartilage of the first rib is seen to be mottled, as though it were undergoing ossification, although the patient was only twenty-one years of age. On the left side is a still more peculiar condition. The first rib, instead of curving forward from the spine, as does the right, changes its direction at its articulation with the transverse process and thence runs almost in a straight line downward and outward. The tip of it is lost at the point where the first rib and the clavicle cross each other. From that point to the sternum there is no rib, but on the sternum, at the point where the two lines B B would meet, is a conical projection from the sternum just below the end of the clavicle. This represents the point at which the first rib would have articulated had it reached the sternum.

When I first examined the patient I felt certain that he had a cervical rib on each side and I was only undeceived by the skiagraph. Palpation seemed clearly to indicate a cervical rib.

II. *Nervous Symptoms.* The nervous symptoms are considerably more frequent than the vascular. About two-thirds of the patients complain of pain and other nervous symptoms, whereas only about one-half suffer from vascular troubles. Commonly the very first symptom is pain, shooting or neuralgic in character, starting usually in the neck and radiating down the arm sometimes as far as the elbow, sometimes all the way to the hand; more commonly on the ulnar than the radial side of the forearm. Sometimes the pain radiates to the head; sometimes to the chest. It often begins in one or more fingers and gradually spreads to the other fingers and then extends up the arm. Muscular exertion, especially hard work, is likely to increase the pain, or sometimes is thought to have caused it. It is sometimes worse in winter than in summer.

Again, the involvement of the nerves is shown by diminished or perverted sensation. The cases in which the sensory phenomena have been the most carefully observed, are that of Dr. Lewellys F. Barker in his own person, and by Bernhard. In one of Dr. Patrick's cases (No. 32), he has kindly allowed me to copy his drawings, showing the disturbance of sensation (Figs. 22 to 25), as have also Mr. Sargent and Dr. Buzzard (Fig. 27). In Patrick's case (No. 32) the anesthesia was so marked that an accidental burn of the forearm was felt but little.

The paresthesia is frequently described as the sensation of pins and needles, formication, tingling, numbness, or "going to sleep." The area usually corresponds with the portion of the arm supplied by the lower cords of the brachial plexus. Moreover, there is either diminution, or, in some cases, entire absence of electrical reaction and in advanced cases the reaction of degeneration is shown. In one case (No. 5) there seems to have been a distinct neuritis, so that when the elbow had been extended to 150° it was impossible to extend it farther on account of the pain.

In Hauswirth's case, handling of the extremity produced such pain that it had to be conducted with the greatest gentleness or even abandoned.

The sympathetic nerve apparently has been involved in four cases. Müller's case showed typical paralysis of the right cervical sympathetic. In Hirsch's case there was profuse sweating and coldness on one side, and dryness of the skin on the other. Gordon attributes the rapidity of the pulse in his case to irritation of the sympathetic. In Schönebeck's case (No. 61 in his series) a patient with bilateral symptoms, the left pupil as well as the left palpebral fissure was larger than the right, and there was facial asymmetry. But in this case the cervical rib was associated with syringomyelia.

Hoarseness. In a few cases (Nos. 2, 6, 16, and 19) there has been hoarseness. This is probably due to some actual affection of the throat and not to involvement of the recurrent laryngeal, which is far removed from the possibility of pressure. In only one case, Borchardt (No. 19), has a laryngeal examination been made. This showed that the right vocal chord was paralyzed. This patient suffered also from syringomyelia.

III. *Vascular Symptoms.* I have already alluded to the marked pulsation in the neck. In Ehrich's case (No. 4), on the contrary, pulsation even in the subclavian was entirely absent and the artery felt like a hard cord, owing presumably to a thrombus in the vessel. In very many cases this is probably due to distal blocking of the vessel. On the other hand, aneurysm has been asserted to exist in a number of cases. (*Vide infra.*)

In a considerable number of patients the pulse at the wrist has been entirely absent and not uncommonly it has been absent also in the forearm, the arm, and the axilla. This, doubtless, is due to a thrombus. As a result of the ischemia the hand and often the arm become cold and blue, or more frequently blanched, sometimes even suggesting possible Raynaud's disease. The blood returns very slowly to the part after brief pressure on the tissues. Generally, even after operation has removed the obstruction to the circulation, the pulse fails to return and the obstruction is permanent. In Babcock's case, and partially in my own, the pulse returned after operation, but this is very rare. Yet the collateral circulation usually is partly established so that gangrene (*vide infra*) is unusual. The thrombus is apt to extend somewhat slowly upward to the subclavian, and even that vessel may be involved. I know of no case in which it has progressed beyond this point. In the case of C. L. F. (*vide supra*) it finally involved the subclavian on both sides, but without apparent injury to the vitality of the arms. In this case there was no cervical rib.

Gangrene. In the cases of Babcock, Gordon, and my own in my table, and in those of Cooper, Hodgson, and Gould, owing to the arrest of the circulation, gangrene of the tips of the fingers has followed. Grisson's case was on the verge of gangrene. But in other cases, even when the pulse has been obliterated as high as the brachial and even the subclavian, the re-established circulation has prevented this misfortune. The gangrene has never extended in any case that I have found beyond the first phalanx, and usually is limited to the tips. After operation, or without it, the parts gradually mummify, drop off, and the resulting ulcer heals. It is worthy of note, that of these seven cases two of the six, in which the age is stated, occurred in patients nineteen and twenty-two years of age; the others ranged from thirty-two to sixty-three years.

The effect on the pulse of varying the posture of the arm has been quite marked in a number of cases. On raising the arm the pulse returned, and in addition to this, apparently from the relief of pressure, the pain would diminish. Sometimes a deep inspiration by raising the rib in the neck would produce marked influence upon the volume of the pulse, and in one unique case (No. 4), at the height of inspiration, the pulse disappeared entirely.

Œdema is exceedingly rare. Like Murphy, who says "that in looking over the clinical records of these cases, he was struck by the absence of œdema as a symptom," I, too, have been much impressed with its absence. Yet there are four cases in which there seems to have been a possible partial involvement of the vein. In Gordon's case, two months after the onset of the symptoms, there was swelling of the hand, but no pitting on pressure. The artery was obliterated from the middle of the arm down and gangrene followed. In Hauswirth's case, he expressly states that "the hand and forearm were swollen, blue, and cold;" and Berg, at the operation on his case, found "the veins of the suprascapular and transverse cervical systems considerably enlarged and required double deligation." In the case of de Quervain, before he saw his patient, a soldier, the physician calling for him noted that there was some swelling and redness of the skin. When de Quervain saw him about four months later, he said that on the affected side "the veins of the back of the hand were fuller than upon the other side." In Thorburn's patient, the hand was cold, and there was a very slight œdema "which may have been due to pressure upon the subclavian vein."

In none of these cases is pressure upon the vein more than suspected, and I think it extremely doubtful whether the swelling or the slight œdema had any relation to the subclavian vein.

Murphy explains this absence of œdema "by the fact that the scalenus anticus is attached to the first rib and lies between the artery and vein so that the latter, situated in front of the muscle, has nothing to hold it firm for counterpressure." When the scalenus anticus is attached to the cervical rib, the vein lies in front of the muscle and the artery behind it.

Aneurysm. In ten cases (Poland, Adams, Stiffler, Murphy, Ehrich (Karg), Fischer, Braun, Pancoast, Grisson, and Conner), and a few other more doubtful cases, the symptoms suggested an aneurysm. None was found, at operation, in Pancoast's case, and only flattening of the artery in Murphy's. Conner says that the pulsation "suggested aneurysm." There is no question that at operation the artery has been found enlarged by some observers (Ehrich, Murphy, Keen) (Fig. 4), and at the necropsy in Adams' case a cylindrical aneurysm was actually found extending from the outer border of the scalenus anticus to the brachial.

But the curious fact is that in these cases, as in my own and others, the dilatation of the vessel was external or *distal* to the point where the artery passed over the cervical rib and distal to the point where it passed behind the scalenus anticus muscle. It is difficult to account for the dilatation distal to the point of pressure or constriction. Possibly it may be due to resistance caused by the still more distal blocking of the artery.

Murphy has made a special point that in the inspiratory movements of the thorax the artery is caught between the rib and the scalenus anticus, and he thus explains the aneurysm.

Even granting, as is probable, that the artery is pinched between the muscle and the bone at this point, this obstacle to the circulation should produce a dilatation *proximal* to the point of constriction and not distal, just as in the case of obstruction of the intestines from a band the bowel is distended proximal to the band and shrunken on the farther side. The observation in my own case was very carefully made and I am absolutely positive of the small caliber up to the point at which the artery emerged from behind the scalenus anticus, and its caliber was nearly doubled from there down to the level of the clavicle. In Murphy's case (No. 31) the artist has represented what, at first sight, seems to be an aneurysm of large size distal to the scalenus. Dr. Murphy's text describing the case shows that there was no aneurysm, but that the artery "was somewhat enlarged" and in five weeks "had contracted to its usual size." The illustration, Dr. Murphy informs me, was intended to show only flattening of the artery. In Poland's and Fischer's cases compression produced early consolidation in the artery and resulted in cure and a return to work.

Usually the pulsation is visible as well as palpable and is very marked. It is occasionally noted that it was "expansile." Bruit or thrill, one or both, are often present, the latter less frequently than the former. The bruit, however, is not propagated. Neither the thrill nor the bruit seem usually to be of the true aneurysmal character. Moreover, the "aneurysm" rarely increases in size or symptoms. Under pressure, or more often without any treatment, it either remains as before, or lessens, and may disappear. In no case has the vessel been ligated; a very wise abstinence.

Another curious case is that of Ehrich (No. 4), in which though at operation the walls of the artery are described as apparently sound the aneurysm developed *after* operation. This may have been due to the loss of support of the bone and other tissues around the artery and to the injury to the walls of the artery by the constant respiratory compression between the two ribs.

On the whole, the evidence of true aneurysm in most of the cases in which it has been reported, are, to my mind, by no means always convincing. Usually the diagnosis has been based on the strong pulsation, sometimes with bruit and thrill. In a few cases, as in my own, the artery has been found, at operation, moderately enlarged, or in one case (Murphy's) flattened. In several cases operation has, therefore, disproved the presence of the supposed aneurysm. The postmortem in Adams' case disclosed a cylindrical aneurysm. Bearing upon the history of pressure or tension of the artery as it crosses the cervical rib, it is significant in Fischer's (Braun's) case that when the arm hung *down* (pressure or tension) bruit was present, and when it was held up (relief of pressure) the bruit disappeared.

That these aneurysms should have undergone spontaneous cure without treatment, other than bandaging and pressure, or removal of the rib, is certainly sufficient to make one suspicious of there ever having been a true aneurysm, except in a very few cases.

In a letter to Prof. Turner, Paget very suggestively says: "In each case the imitation of aneurysm was close enough to deceive an unwary surgeon; but to one who examines closely, and has in his mind what the case may be, the mistake seems scarcely possible, so long as the artery is healthy."

Thrombosis. Allied to the question of aneurysm is that of thrombosis. Here we are on much firmer ground; for that there is a constriction of the artery and an interference with the blood current which might be followed by coagulation there is no doubt. Thrombosis has been recorded by Hodgson, Cooper, Adams, Coote, Braun, (Fischer), Ehrich (Madelung), Gordon, Keen, Fischer, and Grisson.

The extent of the thrombosis varies somewhat, but may involve the arteries of the forearm, not uncommonly of the arm, the axillary, and even the subclavian. Occasionally the progress of the thrombosis has been followed almost day by day, from wrist to axilla. Pulsation in the artery is then, of course, absent and the artery itself is felt as a hard solid cord. After removal of the rib, it is rare for the pulsation to return (Babcock, Keen). The collateral circulation, as would naturally be expected, is established after a time, but is often so imperfect that the skin is very cold and white or blue; and after pressure the color due to the restored circulation returns very slowly. Gangrene of the finger-tips has been considered.

IV. *Muscular Symptoms.* The muscles of the arm on the affected side usually undergo gradual wasting, so that the circumference of the arm and forearm will be from 1 cm. to 4 cm. less than the opposite one. This wasting is especially marked in the thenar and hypothenar muscles, not uncommonly also in the interossei; often the muscles of the forearm are involved. Occasionally the loss of power extends to the muscles of the shoulder and the chest. Along with this there is markedly lessened usefulness of the arm and especially of the hand in the more delicate movements required in sewing, writing, etc. The arm becomes easily tired and the fingers stiff. If the work is at all severe it has to be given up. It will be noticed that the muscles as well as the sensory area involved point to the lower cords of the brachial plexus and not to the upper.

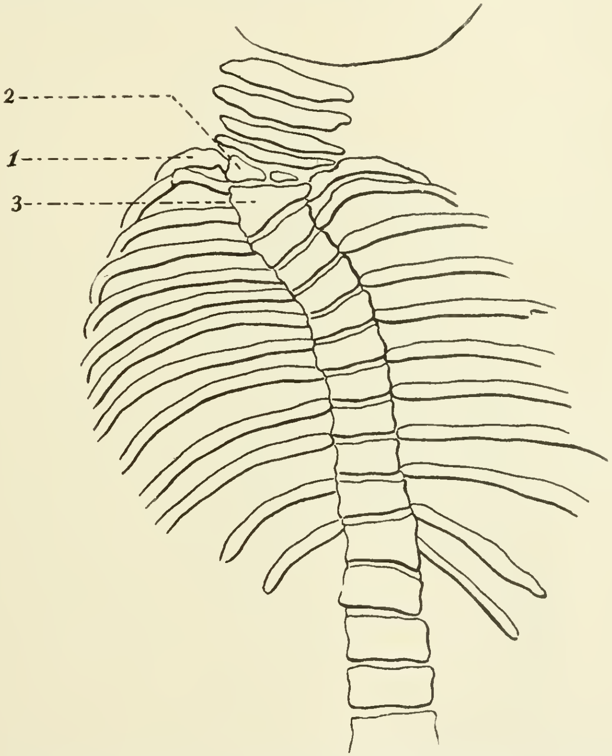


Fig. 18.—Scoliosis with cervical rib; child, aged eighteen months. Note 1, cervical rib attached to one portion of 2, the intercalated body of a cervical vertebra in two pieces. 3. Wedge-shaped body of the first dorsal vertebra bearing two ribs. (Drehmann, Fig. 2.)

Dysphagia. In the cases of Bernhardt, Ranzi, and Tilmann, dysphagia was also noticed. This, most likely, was hysterical, since there is no evidence of there being any direct pressure or interference with the function of the œsophagus. Yet this symptom was so severe in a case seen by Kiliani as to suggest even cancer of the œsophagus. Both he and Lilienthal agreed that it was due to muscular spasm.

Scoliosis. In Schönebeck's sixty-one collected cases (1905) scoliosis existed in twenty-two. In several of the cases operated upon, scoliosis is also recorded. This, of course, is of special interest to the orthopedic surgeon. The convexity is usually toward the side of the cervical rib, or if a rib is developed on both sides, the convexity is toward the side of the larger rib. The deflection extends from about the fifth cervical to the fourth dorsal vertebra. It usually is attended also with a high scapula on the convex side.

In 1902, Garrè called attention to scoliosis in connection with cervical rib. Before this Drehmann had reported four such cases, but had not at that time the advantage of the investigation by the *x*-rays.

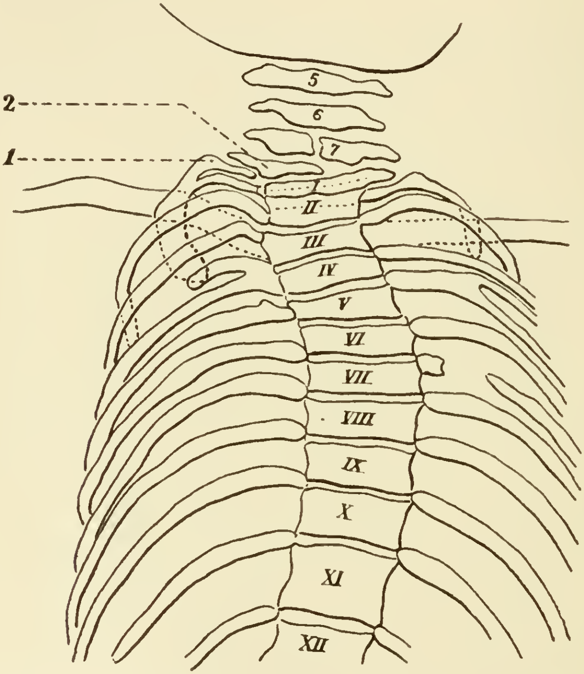


Fig. 19.—Scoliosis with cervical rib. Boy, aged twelve years. Note 7, the body of the seventh cervical vertebra in two pieces. Between it and I the first dorsal vertebra is 2, an intercalated fragmentary body of a vertebra carrying 1, a rudimentary cervical rib, which quickly fuses with the first dorsal rib. Note also several other abnormalities of the ribs on both sides. (Drehmann, Fig 6.)

In 1906, at the Congress of the German Orthopedic Association, Drehmann returned anew to this subject and presented the most important paper on this form of scoliosis which has yet appeared. Moreover, the article is illustrated with reproductions of skiagraphs of his cases, and what is most useful, outline sketches indicating what may be seen in the skiagraphs. It requires a little imagination, I confess, to identify all the parts of the outline drawings in the skiagraphs, but enough is seen to convince one that in all probability the sketches are faithful to what is seen in the originals, which, of course, are very much clearer than the reproductions, excellent as they are.

In one child, aged eighteen months, with wryneck, but without rickets, the skiagraph showed (Fig. 18) a marked scoliosis in the cervicodorsal region. "On the convex side there is a fully developed cervical rib which stands in relation with a supernumerary vertebra. This vertebra is divided into two parts: a large wedge-shaped portion, which carries the rib, and a small rudiment on the concave side. On the convex side of the scoliosis the first dorsal vertebra carries two ribs. It is made up of two parts; the upper piece corresponds to the normal vertebra, under which is placed also a wedge-shaped vertebra, which also carries a rib." It will be observed also that on the right side the twelfth vertebra carries two ribs.

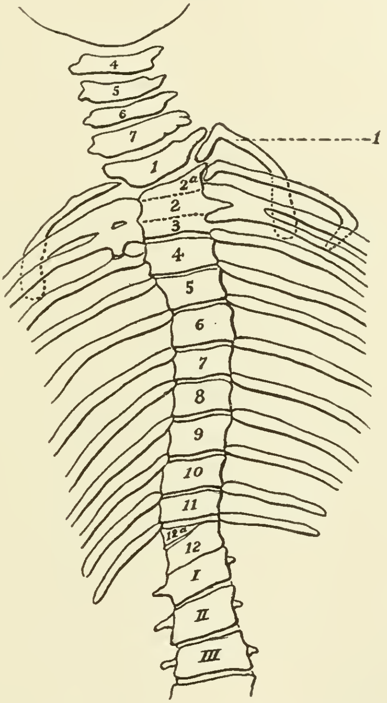


Fig. 20.—Scoliosis with cervical ribs. Girl, aged fourteen years. Between the bodies of the first and second dorsal vertebræ is a wedge-shaped supernumerary body, 2*a*, fused with the bodies of the second and third dorsal vertebræ. This rudiment carries a cervical rib, 1. Several other anomalies of the ribs and the vertebral bodies are to be seen. (Drehmann, Fig. 8.)

In another case, a boy, aged twelve years, the skiagraph shows "A division of the seventh cervical vertebra, and between it and the first dorsal vertebra a wedge-shaped vertebral rudiment is intercalated, which on the convex side carries a cervical rib. This cervical rib unites with the first rib (Fig. 19). The first and second dorsal vertebræ cannot be differentiated. They seem to be blended with each other. The fourth rib on this side is also rudimentary. On the opposite side the fourth rib bifurcates, and it seems as if on both sides the fourth and fifth ribs are united. The seventh rib also shows a defect on the right side."

Another case (Fig. 20) shows several anomalies in the ribs and the vertebral bodies, as noted in the legend.

Several of his figures also show some remarkable deviations from the normal in the lumbar vertebræ, as well as in the cervicodorsal.

He calls attention to the fact that up to the present time congenital scoliosis has been held as an extreme rarity. He believes this to be an error and urges methodical examination by the *x*-rays.

The cases he describes are divided into two classes: the first group are those in which the supernumerary rib belongs to a corresponding rudimentary intercalated vertebra; the second group, those cases in which a true cervical rib exists, without any intercalated vertebral fragment.

As to treatment, he believes that extirpation of the supernumerary rib in these cases is entirely useless, since they ought not to produce any pressure symptoms on the plexus.

Cause of the Symptoms. Borchardt believes that only about 5 to 10 per cent. of the cases of a cervical rib are followed by any symptoms. Whether this is at all an accurate percentage is rather unimportant; it is certain that the vast majority of cases, in which this anomaly exists, have no symptoms whatever which can be attributed to the presence of the rib. There must be, therefore, something exceptional in the cases which show symptoms different from the majority. I am inclined to believe that in Laplace's case the symptoms were due to the severe trauma to the shoulder and not to the presence of the cervical rib.

Injury. In a certain number of cases there can be no question that injury, direct or indirect, is responsible for awakening the symptoms. Thus, in cases Nos. 2, 23, and 33 the patient had a fall. In Poland's case the drawing of a cork was attended with a feeling as if something had been torn loose in the neck. In Stiffler's case, lifting of the arm was attended with a feeling of a tear in the neck and was assigned as the cause of the resulting aneurysm. In three cases (Nos. 4, 7, and 15) pressure of the rifle at "shoulder arms" or the pressure of the straps of the knapsack were clearly the cause of the trouble, and Brewer's patient was a peddler. Other occupations, such as that of a seamstress, sewing base-balls, paring potatoes for two hours (Marburg), writing, piano playing, etc., seem to have had a definite influence.

In consequence of the protrusion of the end of the rib, there is more danger of injury than where the parts are normal. In

Williams', Borchardt's (No. 18), and Starr's cases, even so slight a constant but repeated injury as the pressure of the collar seems pretty clearly to have evoked the earliest symptoms.

Ill Health. It is to be noted that in a very considerable proportion of the patients ill health preceded the first symptom. Repeatedly, anemia, chlorosis, general ill health. or some debilitating disease is noted. This may have been a factor in arousing the trouble by the wasting of the fatty tissues in the neck itself besides its general predisposing influence. In Borchardt's case (No. 20) diphtheria seemed to be the cause of the trouble; possibly by the direct toxic effect.

Pressure, Constriction, and Sharp Bending of the Artery. Pressure and constriction of the artery may arise in two ways. I have already alluded to the view that Murphy has particularly advocated that there is marked compression of the artery in the angle between the rib and the scalenus anticus. That this is true I think there is no doubt. In Weissenstein's case, dissection shows excellently how this is brought about (Fig. 9; see also Fig. 4). Halbertsma and Luschka have advocated the view that the trouble is due to direct pressure of the rib on the artery, especially in inspiratory movements in which the cervical rib would lift the artery by muscular effort. At the rate of sixteen inspirations in the minute, an upward excursion of the rib occurs nearly 1000 times in an hour and about 23,000 times in a day. If continued day after day for years, even this slight upward pressure in lifting the artery might easily produce such an obstacle to the circulation and such a possible effect upon the arterial walls as to be followed by a diminution in the volume and force of the pulse and not uncommonly by thrombosis. Gruber believes that there can be no such tension of the artery from upward pressure, and Sue insists that if the cage of the chest is developed vertically to a greater extent than is normal the arteries would also develop *pari passu* in length. This view of co-ordinate development of the chest and the bloodvessels seems very plausible, especially in view of the fact that only a small percentage of cases, in which cervical rib exists, do show any symptoms. But to my mind, the argument that pressure and its results really exist in the cases with symptoms is valid for the following reasons:

1. As a matter of fact, at operation a sharp bend in the artery has been especially noted by Ranzi and was marked in my own case. In Borchardt's case (No. 18) traction on the hand downward was seen to cause complete compression of the artery between the scalenus and the rib, and in de Quervain's case, certain movements of the shoulder compressed the artery between the rib and the clavicle.

2. In a number of cases the moment that the rib has been removed the artery has sunk down into the soft tissues of the neck, showing the relief of tension, and the symptoms have quickly disappeared.

3. Murphy writes me that in his first case (No. 31) the artery was *flattened.*

4. In Kammerer's case, the pulse completely disappeared on deep inspiration and in others it was weakened.

5. It should also be pointed out that the argument has been applied only to the artery. The nervous symptoms, due to the lesion of the brachial plexus, are much more frequent than the circulatory symptoms. As to the nerves, the argument of Sue entirely fails, because the brachial plexus arises in the neck *above* the level of the cervical rib and the course of the plexus is from above downward and outward. Angulation cannot then be assigned as the cause, as in the case of the artery; but the upward pressure of a longer cage of the thorax certainly exists and this upward pressure is constantly repeated, as in the case of the artery at every inspiration. Coote, in the first case ever operated on, expressly states that "the nerves were *flattened* upon the upper surface of the rib." Warren notes that the brachial plexus "had been somewhat *stretched* by the arched position of the bone." Removal of the bone relaxed the plexus. In my own case the same phenomena were observed. In Thomas and Cushing's case direct "compression" of the lower cord of the plexus "by a dense fibrous band which passed from the tip of the rudimentary rib under the plexus to its point of attachment on the first thoracic rib" was found. This band represented the continuation forward of the imperfectly developed cervical rib.

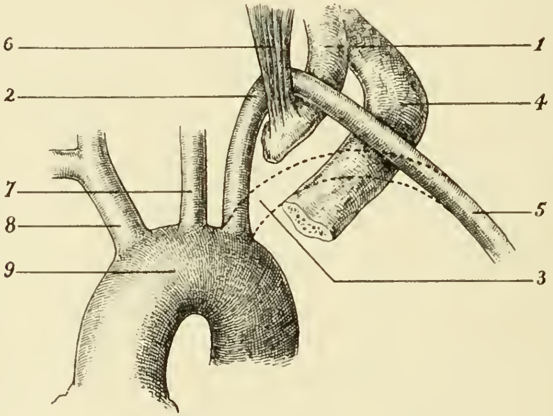


FIG. 21.—Scheme to show the changed course and the consequent angulation of the subclavian artery when it passes over a cervical rib. The dotted line (3) represents the normal gentle curve of the artery. 1. Cervical rib. 2. Subclavian artery passing over the cervical rib. Note its high position, its angulation, and the likelihood of pressure by the scalenus anticus 6. 4. First dorsal rib. 5. Axillary artery. 7. Left carotid. 8. Innominate. 9. Aortic arch.

6. Moreover, as in the case of the artery, the removal of the offend-

ing rib almost always has been followed by sinking downward of the plexus and relief of the nervous symptoms.

7. Again, the *lower cords* of the plexus are much more frequently involved than the upper. This is consonant with the theory that it is the upward pressure of the cervical rib lifting the lower, rather than the more distant upper cords which produces the lesion. Naturally, the upper cords would be the most likely to escape. The artery would not show this so well, since its walls are elastic and its contents fluid.

Angulation. The subclavian artery, arising either on the left side, directly from the aorta, or on the right side, from the innominate as it passes outward in a gentle curve to the axilla, rises normally into the neck a little above the upper border of the clavicle. In Fig. 21 the dotted line shows its normal course. In cases of cervical rib the artery usually rises in the neck from 3.5 to 5 cm. or more above the level of the clavicle and must bend acutely downward and outward to reach the axilla, as shown in Fig. 21. This shows the difference between the normal course of the subclavian artery and by contrast its course when it runs over a cervical rib. It shows also the *necessary* sharp bend or angulation in the artery when a cervical rib is present. See also Figs. 9, 12, 13, 14. and 15. In almost every case it is noted that the pulsation of the subclavian artery was felt or seen, or both, far above the level of the clavicle, instead of being just behind that bone or barely above its level. This sharp bend or angulation of the artery, in addition to tension or pressure, I believe to be of considerable influence in producing the vascular symptoms.

How can we Account for the late Appearance of the Symptoms? The age at which symptoms have been noticed in the 42 cases operated on was as follows: Under fifteen, 6, that is, at seven, nine, ten, twelve, thirteen, and fourteen years; fifteen to twenty, 15; twenty-one to thirty, 8; thirty-one to forty, 11; sixty-two, 1; not stated, 1.

It will be seen that in a large majority of the cases the symptoms do not arise until after fifteen years of age, and in some comparatively late in life. Murphy is disposed to ascribe the outbreak at puberty or later to the growth of the rib occurring chiefly between the cartilage of the anterior end and the bone. Naturally, with growth in size from childhood to adult life, all the bones grow in length, and a cervical rib would be no exception. It seems to me that in the cases in which the symptoms appear late in life, as in my own case at sixty-two years of age, and Rutkowski's at fifty-nine, and others not in my table about the same age, this is not due to any one but to a number of factors. For example, arteriosclerosis would have some influence as the arteries begin to lose their elasticity, and if they are subject to any unusual pressure between a cervical rib and the scalenus anticus or by the repeated lifting action of the rib, changes in the wall of the vessel would be likely to occur which might easily lead to retardation and even to thrombosis. I have seen a case of senile gangrene of both feet, from calcareous arteries, at forty-five years. Moreover, as we get older, the muscles become more flabby and lose their tone, so that the shoulders sink to some extent. This would naturally increase the angulation of the artery where it passes over the cervical rib and also increase the pressure whether from the rib or from the scalenus anticus.

Again, if the rib itself is long, and especially if it reaches well forward on the first rib or even to the sternum, the space between the clavicle and the cervical rib would be much narrower than it would be in cases without a cervical rib. This may produce an increase of pressure, especially in any movements of the shoulders backward which might be involved in daily labor. In Schnitzler's case the anterior end of the abnormal rib approached so close to the clavicle that it acted as a fulcrum, so that when the shoulders were thrust backward the inner end of the clavicle was dislocated. Garrè also has called attention to bending of the clavicle at the junction of the middle and outer thirds by reason of similar pressure.

Babcock considers "that the symptoms result from the increased size or the greater fixity of the rib," and alludes to the fact that the ossification in the transverse process does not begin till puberty and is not complete till about twenty-five years. Moreover, in the head and tubercle of the normal rib ossification begins at fifteen years and is completed at about twenty-three years. "It is obvious, therefore, that a cervical rib will tend to become progressively larger, more fixed, and have greater tendency to cause pressure symptoms between the ages of fifteen and twenty-five years."

Treatment. The majority of cases of cervical rib require no treatment whatever. For those who are suffering pain, which is the most frequent reason leading the patient to consult the surgeon, it is right that medical treatment should first be tried and that we should not rush heedlessly to a surgical operation, unless the conditions warrant it. For example, cases in which the patient is suffering *severe* pain, or when gangrene of the tips of the fingers is either present or impending, admit of no delay. But inasmuch as there are several cases on record in which the patient has not been bettered by operation because of a concomitant progressive central nervous disease, such as syringomyelia, it would be right, if delay is possible, to test the effect of medical means until sufficient time has shown that this will do no good, or that the case is not simply one of cervical rib, but of a much graver condition of the central nervous system. These medical means are first of all rest, combined, it may be, with massage, liniments, electricity, and douches. If the patient's general health is poor, as is so often the case, of course this should be attended to and suitable hygienic and therapeutic measures should be instituted. If the case is supposed to be one of aneurysm, pressure by a pad and bandage may be applied. While remedies to quiet the nervous system and possibly hypnotics for the purpose of procuring sleep and relieving pain may be desirable in some instances, yet their prolonged use, and still more the use of opium in any of its forms, is certainly to be condemned. If the case is severe enough to require large doses of morphine with any frequency, it is severe enough to require operation.

On the other hand, delay that may permit thrombosis to occur and, especially, to progress even to gangrene, or to allow the muscles, because of the interference with their vascular or nervous supply, to atrophy and become useless, and the joints to become partially ankylosed is even more to be condemned. Each case must be judged upon its own merits and by its own symptoms and dangers.

Operative Treatment. The rapid increase in the number of operations as the condition has been more readily diagnosticated, and especially since the x-rays were discovered, in 1895, is remarkable. In the thirty-five years from 1861 to 1895 only eight operations are recorded. In the five years from 1896 to 1900 nine more are recorded. From 1901 to October, 1906, inclusive, that is, less than six years, twenty-five cases have been published.

The most important fact as to the whole series of operations is that the *mortality has been absolutely nil.* Not a single patient died as a result of the operation nor was a single one seriously ill, excepting those few cases in which other diseases of the nervous system, especially syringomyelia, were present. In a few the result has not been all that could be wished, but in the great majority it has been most satisfactory. In only three cases has there been recurrence, necessitating a second operation in two. In Beck's case a subperiosteal resection was followed by reproduction of a centimeter of the rib and recurrence of the symptoms. In Lilienthal's the symptoms were thought to be due to the cicatrix. No second operation was done. In Marburg's case the symptoms persisted and a second operation was done, but the persistent symptoms were probably due to the syringomyelia. In no case has a bilateral operation been required.

Operation. Three different incisions have been used: First, horizontal or nearly so, that is, parallel with the clavicle, similar to that by which we ligate the subclavian artery. Second, vertical or obliquely vertical at a suitable position and angle between the sternocleidomastoid and the trapezius muscles. Third, a combination of these two, either by an incision, one limb of which is parallel with the clavicle or nearly so, and the other more or less nearly vertical, meeting the horizontal one at the anterior end, or at some point in its course. The last one was the incision I adopted. It is, I think, a matter of indifference which of these methods is adopted. Each operator will select the one which is best, in view of the development of the rib and the depth to which the dissection must be carried. In a long thin neck the vertical incision would be the best; in a short stout neck a combined incision. When the patient's neck is of medium length and there is moderately thick subcutaneous fatty tissue a horizontal incision would very probably give sufficiently wide access. The main thing, no matter what incision is adopted, is to have plenty of "elbow-room."

Misled in his diagnosis, thinking the case one of malignant disease of the vertebræ, Brewer divided the clavicle. No harm came of it. But this is not a necessary step. In Williams' case it was considered, but rejected.

Exposure of the Tissues. Sometimes the superficial vessels in the neck are in the way either by their size or position. In these cases, whether artery or vein, they should be doubly ligated and divided. By blunt dissection we then approach the tumor formed by the rib and expose the brachial plexus and the artery. Sometimes the one or the other may not be seen. The vein is rarely seen, but its presence should always be remembered lest inadvertently it may be wounded. So, too, injury to the phrenic nerve must be carefully avoided.

The rib is now exposed by blunt dissection both anteriorly and posteriorly as far as is either possible or necessary. Very commonly if the rib is of considerable length, the scalenus anticus and very possibly the medius and posticus may be inserted into it. In such case the muscle must be separated by scissors or knife from the bone.

In isolating the rib, one of the chief dangers is the dome of the pleura. In my own case I did not see it. Although the patient was rather thin, there was sufficient fatty tissue in the neck to obscure it and it never came into the operative field. In other cases it has not only been seen, but sometimes it has been very difficult, or even impossible, to avoid injuring it. In five cases it has been torn to a greater or lesser extent (Planet, Patterson, de Quervain, Brewer, and Tilmann). In no case, however, has this proved to be a serious complication. The moment that it occurs the surgeon is aware of it by the noise of inspiration and expiration. The opening should be instantly closed by the finger and then by packing with iodoform gauze. A slight local pleurisy may follow, but in most cases only a passing trouble has resulted from this accident.

The rib is then removed as far as necessary both backward and forward, either by the chisel, the Gigli or other saw, or the bone forceps, the stump at each end being rounded off by the rongeur forceps. It is important that the stumps, especially the anterior one, whether this is the anterior end of the rib or an exostosis on the first rib, should be sufficiently removed, lest, as in Gordon's case, the subclavian is "slung" over the projection and thus still seriously interfere with the circulation. (Fig. 5 shows the stump in my own case.) In Tilmann's case it was a cause of annoying pain. If the anterior extremity is free, it facilitates removal. If the rib is attached to the first dorsal rib, and especially if this is a bony attachment, it makes its removal somewhat more difficult. In this manœuver the pleura, of course, must be very carefully protected from injury.

While it is generally recommended to remove the periosteum with the bone, this was not done in Borchardt's case (No. 11), and there was no reproduction of the rib as shown by a skiagraph four years later. Rafin also followed this method. Israel even advocates subperiosteal resection. But, as removal of the periosteum does not materially add to the difficulty of the operation, and as Beck's case showed that reproduction did actually take place and required a second operation, the periosteum should always be removed with the rib.

In uncovering and removing the rib, the brachial plexus should be drawn outward and upward and the artery downward and inward. So far as the artery is concerned, its walls are usually so strong that there is little danger of doing it injury. No surgeon capable of such an operation would probably injure the artery. There is no such instance on record. With the plexus, however, it is different. In order to uncover and remove the bone, several operators have been obliged to drag upon the plexus so seriously as to produce severe pain for several days after operation, and in some cases partial or complete paralysis of the arm (Gordon, Borchardt (No. 18), Ranzi, Marburg, and Patterson). In the great majority of cases, both the pain and the paresis or paralysis have only been temporary, but in a few cases they have been a very serious and more or less permanent sequel. It warns us, therefore, to be on our guard and to handle the plexus with as great gentleness as possible.

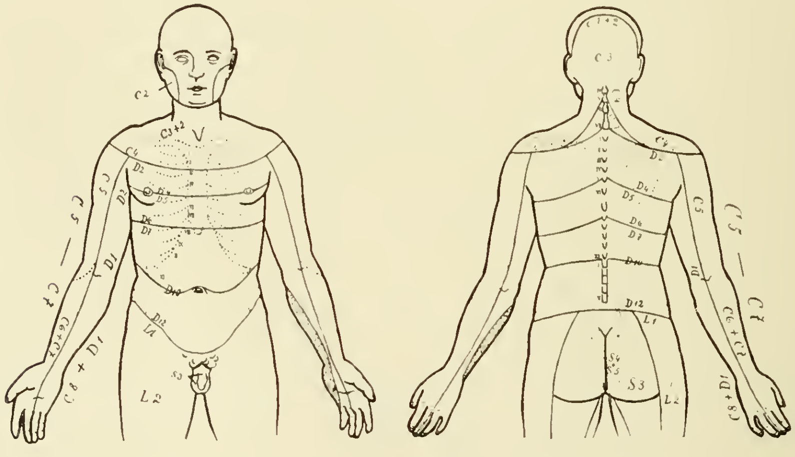
As soon as the rib is removed and the ends smoothed off the artery and plexus sink into the gap felt by the resection, and no further attention need be paid to them. The wound is closed in the usual manner, with or without drainage as may be thought desirable. de Quervain warns against drainage in case the pleura is torn on account of the possible danger of emphysema. Usually the wound is entirely healed in the course of a few days with but little if any reaction.

The after treatment should consist of massage of the arm, electricity, and passive and active movements in order to restore the usefulness of the hand and arm.

Case 32. Hugh T. Patrick and William E. Morgan (personal communication). Woman, aged twenty, no occupation, first seen September 24, 1904. Three or four days, or possibly more, before examination the left arm began to ache with an almost constant dull, "dead" pain. About eighteen months before examination she had noticed some disability of the left hand and arm. Found she would drop things, and noticed it was difficult to do up her hair.

TABLE OF 42 CASES OF CERVICAL RIB THAT HAVE BEEN OPERATED ON FROM 1861 TO THE AUTUMN OF 1906, ARRANGED IN CHRONOLOGICAL ORDER.[6]

No.	Author.	Operator.	Date.
1	Coote	Coote	1861
2	Planet	Périer	1889
3	Fischer	Fischer	1891
4	Ehrich	Karg	1891
5	Ehrich	Madelung	1894
6	Tilmann[7]	Bardeleben	1894
7	de Quervain	de Quervain	1895
8	Warren	Warren	1895
9	Czerny	Czerny	1897
10	Grounauer	Grounauer	1897
11	Borchardt	v. Bergmann's Clinic	1897
12	Williams	Williams	1897
13	Thorburn	Thorburn	1898
14	Gordon	Gordon	1899
15	Kiderlen	Schede	1899
16	Ranzi	Friedlander	1899
17	Rafin	Rafin	1899
18	Borchardt	v. Bergmann's clinic	1900
19	Borchardt	v. Bergmann's clinic	1901
20	Borchardt	v. Bergmann's clinic	1901
21	Brewer	Brewer	1901
22	Israel	Israel	1901
23	Kammerer	Kammerer	1901
24	Pancoast	Frazier	1901
25	Ranzi	v. Eiselsberg	1902
26	Riddle	Riddle	1902
27	Dejerine & Armand	?	1903
28	Thomas & Cushing	Cushing	1903
29	Thorburn	Thorburn	1904
30	Marburg	v. Eiselsberg	1904
31	Murphy	Murphy	1904
32	Patrick[8]	Morgan	1904
33	Beck	Beck	1905
34	Babcock	Babcock	1905
35	Hauswirth	Berg	1905
36	Lilienthal	Lilienthal	1905
37	Patrick[8]	Plummer	1905
38	Keen	Keen	1905
39	Murphy	Murphy	1906
40	Sargent & Buzzard[9]	Sargent	1906
41	Laplace[9]	Laplace	1906
42	Patterson[9]	Patterson	1905



FIGS. 22 and 23.—Front and rear views of Patrick's case (No. 32). Tactile anesthesia, with slight hyperalgesia of the left forearm, slight in degree and with ill-defined border.

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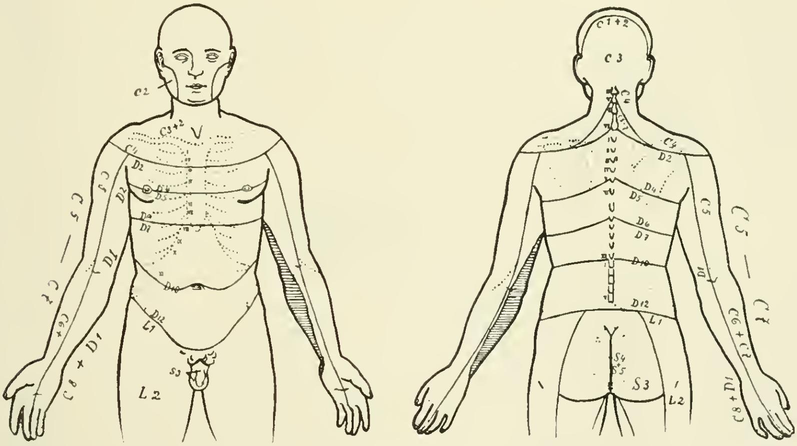
[6] In the Bibliography (arranged alphabetically) the references to all of these cases which have been published will be found. It has not always been clear who was the operator and, therefore, I may have incorrectly stated the name in a few cases. Abstracts of many of the earlier cases are given by Eisendrath, Pilling, and Schönebeck. Of the unpublished cases brief histories are given below. I have to thank the surgeons who have communicated these various cases to me personally.

[7] This case is also reported in Kuester's Inaug. Dissert., Berlin, 1895, and is often referred to as his or as Bardeleben's.

[8] As Dr. Patrick's cases were published in such very brief abstracts, I give here considerably fuller histories, which I owe to his kindness. Cases 38, 41, and 42 are also from notes kindly furnished by the operators.

[9] Not yet published.

After any unusual exertion, pain in the arm was increased; occasionally kept her awake at night. At about the same time she began to notice a numb feeling and paresthesia along the inner border of the left arm, forearm, and hand. Of late she has noticed that the left hand was not so sensitive as the right, and a few days before being examined she burned the ulnar side of the wrist and noticed that it was not normally painful. There was atrophy of the small hand muscles and beginning claw-hand. Measurements showed a difference of ½ inch in the circumference of the hands, ¼ inch in the forearm, and ¾ inch over the biceps. There was slight flattening of the region of the left supraspinatus, slight wasting of the deltoid. In the action of shoving the left scapula "winged" a little more than the right. The grip was slightly weak on the left side and, in grasping strongly, the hand turned slightly toward the radial side. The palpebral fissure was slightly smaller on the left side and in obscure illumination the left pupil was slightly larger than the right. There was slight but distinct thermo-anesthesia along the inner border of the arm and forearm from the wrist (not including the hand and little finger) nearly to the axilla. Along this border there was also some tactile anesthesia, but smaller in area than the thermo-anesthesia (Figs. 22 to 25). There was reaction of degeneration in the small hand muscles.



FIGS. 24 and 25. Front and rear views of Patrick's case (No. 32), same as Figs. 22 and 23. Area of thermo-anesthesia (incomplete) of left arm; border indistinct.

Operation, October 25, 1904, by Dr. William E. Morgan. No particular difficulty was encountered at any stage. The patient made a good recovery from the operation, although she experienced a good deal of pain for the first two or three days. No later history was obtained.

Case 37. Hugh T. Patrick, and S. C. Plummer (personal communication). Woman, aged twenty-four, bookkeeper and stenographer, first seen January 25, 1905. Pain had begun in the right axilla eight years before and had gradually extended down the inner border of the arm, gradually becoming worse. Any over-exertion, such as prolonged writing or piano playing, increased the pain, and she positively affirmed that any undue excitement had the same effect. About four years before being seen, she first noticed disability in the fingers, in trying to pick up a pin. Three years before, loss of power in the thumb had reached such a degree that she could not hold a pencil in the ordinary way, and was forced to hold it between the index and middle fingers. By January, 1905, there was not only weakness in the small hand muscles, but the whole right arm was weaker. The pain seemed to start in the axilla and run down to the elbow and middle of the forearm, along the inner border. She had no pain in the hand or fingers, but occasionally pain seemed to extend from the axilla around to the scapular region. She did not know when the wasting had begun, but when she first noticed it two years before examination it had already become quite marked.

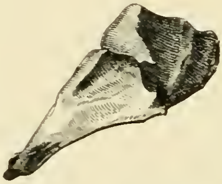


FIG. 26.—Specimen removed in Patrick's Case 37. (Natural size.)

In the history of this case it is rather interesting to note that a diagnosis of writer's cramp had been made by a prominent neurologist, because of the difficulty in writing and the distress caused by it. Measurement showed the right hand, forearm, and arm each $\frac{1}{4}$ inch smaller than the left.

No distinct anesthesia could be made out. The skiagraph showed not only a cervical rib on the right, the affected side, but also one on the left almost as large. As the left rib has caused no symptoms, of course it has been let alone.

Operation, March 16, 1905, by Dr. S. C. Plummer. Incision, three inches long, from clavicle at posterior border of sternocleidomastoid muscle, obliquely upward and backward. Skin and platysma divided, also a part of trapezius and of sternocleidomastoid, as these two muscles came close together, and the portions cut were quite thin. Brachial plexus exposed. It was not necessary to expose the subclavian artery. The posterior belly of the omohyoid muscle was exposed, and drawn forward and downward, along with the plexus and artery. The scalenus medius was laid bare and retracted backward. There were no muscular attachments to the cervical rib. The cervical rib was now freed and cut off at its base with a chisel. A portion of the remainder of the rib was cut away with rongeur forceps. The rib was 1¼ inches long and ⅜ inch wide at its widest part (Fig. 26). Its anterior end was pointed and connected with the first dorsal rib by a slight ligamentous band. No injury to pleura. Catgut sutures in muscles. Silkworm-gut in skin. No drainage.

After the operation she suffered very considerable pain above the clavicle, about the site of the operation, not under the arm and along its inner border, as heretofore. This pain continued for about six weeks without much change, when it gradually diminished. However, as soon as the patient began to use the arm, such use caused pain in the scapular region, and if the exercise was continued the whole arm began to ache. There has been constant but very slow improvement. The patient was last seen on August 10, 1906, and while the pain and tenderness above the scapula had almost entirely disappeared and she had no pain in the arm when quiet, exertion of almost any kind, if continued for more than twenty or thirty minutes, caused first discomfort, and then a decided ache. As regards atrophy, strength, and function of the hand, it could not be said that there had been any improvement. On the whole, the results of operation were disappointing, although it is reasonable to suppose that improvement will continue for a long time.

Case 38. Francis D. Patterson (personal communication). Woman, aged thirty-five, had suffered from children's diseases; diphtheria at fifteen; quinsy at twenty-one; malaria at twenty-four; rheumatism at thirty-one; typhoid at thirty-four. At twenty-eight she was operated on for an ovarian abscess.

July 19, 1905, at thirty-five years of age, she noticed a swelling on the right side of the neck, with pain in the distribution of the brachial plexus. A cervical rib was diagnosticated. She was operated upon August 2, 1905, by a vertical incision along the posterior border of the sternocleidomastoid. The pleura was punctured and a late pneumothorax developed. Uninterrupted recovery took place, but a pressure paralysis followed, probably as a result of the forcible retraction of the brachial plexus. All the earlier symptoms, caused by the cervical rib, had steadily improved up to January 6, 1906.

Dr. Rhein examined the patient October 26, 1905. There was complete paralysis of all the muscles below the elbow, except that there were feeble pronation and supination. Flexion and extension of the fingers absolutely lost; deltoid unaffected, but pectoral muscles weak; latissimus dorsi, supraspinati and infraspinati intact; biceps and triceps, especially the latter, very weak. Marked tenderness along the nerve trunks. Straightening of the fingers caused great pain. No loss of sensation either to pin-point, heat or cold.

There was some hyperesthesia of the left hand. Faradic contractility was present in all of the muscles, but diminished in the extensors, flexors, triceps, and biceps; the deltoid was normal. There was reaction of degeneration in biceps and triceps, flexors, in the forearm and the small muscles of the hand. The cathodal closures were greater than the anodal in the extensors of the forearm. The prognosis was good.

Case 41. Laplace (personal communication). G. H., aged eighteen, first seen December 4, 1905. On October 17, 1905, while looking out of the window of a train, was frightened by another train passing by, and hurriedly drew his head in, knocking his right shoulder against the window. He was unconscious for about fifteen minutes. When he awoke, he was unable to raise his right arm. Was treated for six weeks by his family physician, without improvement.

Physical Examination. December 4. The patient, though only eighteen, is six feet in height; is quite thin. Has grown very rapidly during the last two or three years. The right shoulder seems especially emaciated. He is unable to raise the right arm. There is considerably atrophy of the deltoid, biceps, and triceps. He is, however, able to flex the forearm and retains the power of pronation and supination. Sensation has remained about normal. Says that he is getting worse since the accident. Considerable bulging was noticed on the right side of the neck. On pressure it was found quite hard and gave considerable pain. The presence of a cervical rib was then suspected. An *x*-ray photograph revealed a cervical rib on both sides, though better marked on the right.

Operation, January 25, 1906. A curved incision was made in the posterior triangle commencing at the centre of the posterior margin of the sternomastoid, curving backward and downward to the anterior margin of the trapezius and then forward along the clavicle to the sternomastoid again. The flap was raised forward, and the deep facia was opened. The sternomastoid was pulled forward, and the subclavian artery and brachial plexus were defined lying upon the rib. The periosteum was incised and the rib was bared. It was found fixed at both extremities. Below, it seemed to be inserted into the sternoclavicular articulation. About four inches of rib was resected with cutting pliers and carefully shaved down so as not to leave any marked projection or sharp edge, against which the vessels or nerves might be damaged. The periosteum was then resected. The wound healed without trouble, but as yet the paralysis of the circumflex and subscapular nerves has not been relieved. About ½ inch of the rib was left adherent at its anterior insertion, fearing to wound the subclavian artery and vein by working any deeper.

Case 42. Mr. Percy Sargent and Dr. Farquhar Buzzard, of London (personal communication). Woman, aged forty-nine.

For ten years she had complained of inconstant dull aching pain in the ball of the right thumb; worse in evening and after muscular exertion. Three or four years ago numbness in tips of right thumb and forefinger was observed. One year ago atrophy of the ball of the right thumb was noticed, and at the same time she began to have aching pain in the internal aspect of the right forearm and over the deltoid. She had occasional pain above the right clavicle. A year ago the numbness extended to the tip of the middle finger. On admission the only muscle affected was the right abductor pollicis; the thumb movements were scarcely interfered with excepting in abduction. For the sensory area, see diagram (Fig. 27). A skiagraph revealed bilateral cervical ribs, but they could not be definitely felt. The subclavian arteries were raised well above their usual levels.

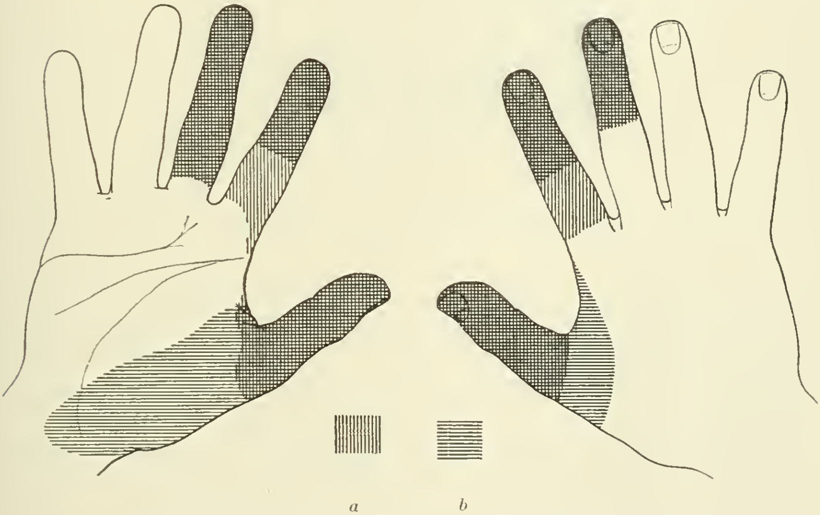


FIG. 27.—Case of Sargent and Buzzard (No. 42). *a*, diminished sense of pain; *b*, diminished tactile sense. The darkest areas represent diminution of both.

Operation, July 17, 1906, by Mr. Sargent. Triangular flap of skin corresponding to posterior cervical triangle; exposure of plexus; complete rib found, articulating with the seventh cervical and attached to the first rib. An angular prominence existed in the middle. "There was no indication that any part of the plexus was pressed or dragged upon in any way."

After operation the whole upper extremity was extremely weak. Sensation was unaffected. September 15, 1906: the arm is recovering, but there is still some wasting of the spinati, deltoid, biceps, and triceps. Sensation is the same as before operation. No definite electrical changes exist in any muscle. October 23, 1906: recovery of muscles of the fifth cervical segment is very slow.

POSTSCRIPT.

After this paper was in print, Dr. J. M. Spellissy, of Philadelphia, reported at the Academy of Surgery of Philadelphia the following case. I owe to his kindness the advance notes of the case before publication and also the photograph of the most complete specimen of cervical rib which I know. It shows a most extraordinarily deep groove for the subclavian artery (Fig. 28).

M. R., aged twenty years, was first seen in May, 1905, for a painful right shoulder, following a fall over two years previously. Examination showed a sternal luxation of the right clavicle and the presence of a right cervical rib. A skiagraph confirmed the diagnosis. There was no cervical rib on the left side. Excision was done by Dr. Spellissy on May 22, 1905. The anterior end of the cervical rib articulated with the first dorsal. This end was disarticulated, and after freeing the under surface of the rib and dissecting the artery loose for 5 cm., with considerable difficulty the artery was displaced over the anterior end of the rib. The rib was then disarticulated from the seventh cervical vertebra. The patient made a good recovery.

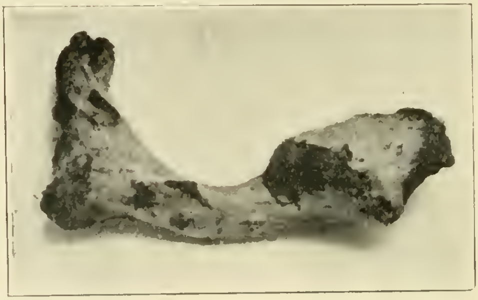


FIG. 28.—Cervical rib (natural size), with very deep groove for the subclavian artery. (Spellissy's case).

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COMPULSORY VACCINATION, ANTIVACCINATION, AND ORGANIZED VACCINATION.

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The antivaccination movement in the United States is becoming widespread and troublesome. The first suit at law against legalized vaccination was brought in 1890.[1] Since then there have been many, and although the decisions almost unanimously favor the health authorities, litigation seems on the increase.

For the last eight years we have been passing through an epidemic of smallpox, and this showed, in strong colors, the need of more vaccination rather than less. I believe that legal regulation is needed, but I also think, and shall try to show, that our present laws are unsatisfactory in their results. If laws are made, they should be so constructed and enforced as to secure the sanction of public opinion. I need not discuss the fiction that in a republic all laws come from the people. This at least is true, that some kinds of laws cannot be enforced unless they have popular acquiescence. Unpopular health laws affecting the person will find difficulties such as arise in the execution of sumptuary laws, like those regulating the use of tobacco and alcoholic beverages. Even among people with despotic or paternal government, with highly organized civil service and active police, vaccination is sometimes defeated. Mothers suck out the virus from their children's arms with great danger to themselves; older subjects wash out their wounds; dishonest physicians give false certificates; venal officials connive. In our own country some legally qualified practitioners of medicine are opposed to vaccination and give certificates for absurd substitutes, such as variolinum, or diluted smallpox virus, given by the mouth.

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[1] Abeel *vs.* Clark, 84 Cal., 226.

What we need, in my opinion, is not statutory compulsion, our first antidote against all evils, from corsets to obnoxious corporations, but an organized and scientific procedure that shall have the confidence and support of a large majority of the people, and shall have no weak spots in any part. Many antivaccinationists are both ignorant and fanatic, but more are merely ignorant. If as serious an effort were made to convert them to vaccination as is now made to turn them the other way, I believe, on the basis of some experience, that they would easily submit. Perhaps the most important objectors are quiet but influential people whose opposition has been aroused by some real or fancied injury wrought by the operation, or by the exaggerated or baseless claims of its advocates. A proper method would turn these into active supporters of general vaccination. Medical writers and health officials too often follow a more arbitrary course, as shown by the following extract from a work on therapeutics excellent in most particulars: "After all is said and done, the final decision of the subject [vaccination] rests with the medical profession; for, in the first place, it alone can properly interpret results." Of course, the final decision does not rest with the medical profession. Even if it did, the results should be capable of being understood by any intelligent and unprejudiced mind.

Let me try to show why we need better vaccination, and how it may be secured. We are now in the later stage of a smallpox epidemic that began soon after the Spanish war, and spread over most of the United States. The exact extent and severity cannot be accurately stated. The most complete data are those of the Public Health and Marine Hospital Service, but owing to the method by which they are collected, these are necessarily imperfect. Taking the figures[2] as given, however, we find the following:

Smallpox Cases and Deaths in the United States.

Years.	Cases.	Deaths.
1899	11,136	553
1900	20,362	819
1901	48,206	1,127
1902	54,014	2,083
1903	40,676	1,448
1904	17,965	709
1905	20,099	354
1906 (first half)	8,542	53

The decline of the epidemic does not mean that the disease is being stamped out finally. It is certain that unless something more is done in the next few years than has been done before, another general epidemic will occur, or numerous local epidemics, just as in the past.

The death-rate has, on the whole, been low, but this also cannot be assumed to be a general or permanent feature. At all times in the history of smallpox mild cases have been observed, and it has been known that a mild case or mild epidemic may give rise to a severe case or a severe epidemic.

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[2] Public Health Reports.

The view has been advanced that the recent mild type is due to hereditary immunity caused by vaccination of parents. There is no proof for this, and it may be a very dangerous idea if followed out. Against it may be said: (1) Vaccination in the United States has not been as widespread as this contention assumes. (2) In some localities where there were severe cases there is no reason to think the parents were relatively or absolutely less protected. So in Cleveland, in 1901, there were 1230 cases of smallpox with 20 deaths, or 1.6 per cent.; in 1902, 1298 cases with 224 deaths, or 17.3 per cent. Vaccination protects the individual, as smallpox does, but the latter does not make immune the children. It is probable that the mild type of the recent epidemic is due to causes partly separate from vaccination and not profitable to discuss at this time.

As the death-rate has been low, the total deaths are hardly enough to give the public much concern on that score. A country that sees its citizens killed off in their best years by typhoid fever without making a move to prevent the disease; that is so indifferent to accidental deaths and homicides in all forms, is not likely to pay much attention to the loss of a few hundred people a year from smallpox. Much more serious to a larger number are the sick. Through these discomfort and inconvenience affect many people, and the mild character of recent smallpox has not greatly lessened the horror traditionally associated with it. It is true that patients in well-managed smallpox hospitals often say they would rather have smallpox than be vaccinated. Many people, especially the ignorant in isolated districts, often have smallpox epidemics without paying particular attention to them. But most people have a fear that is justified by the painful symptoms, dangerous sequels, and high infectiousness of the disease in the unvaccinated. The newspaper account of the treatment of George Francis Train may not have been true, but its publication in journals not of the yellowist kind indicates the general feeling. It also shows how much that is irrational and absurd can be attributed to the medical profession. I have taken the following account from a city paper, where it appeared in double column, with picture, "scare-heads," and display type:

"Make a Row when Disinfected. Stamford, Conn., June 17, 1903. Kicking and shouting and resisting with all his might, 'Citizen' George Francis Train was carried from the smallpox pest-house in Stamford yesterday—and laid in sheets beneath the open sky. Then the physicians made a bath of hot bichloride of mercury, into which the old gentleman was lifted. As soon as his nose was shoved into the bath, Mr. Train burst forth into loud protests against the treatment he had received, bemoaning especially the destruction of the beloved manuscript of his autobiography. Pinned in sheets soaked in bichloride of mercury, he was carried to a closed carriage, in which he was conveyed to the residence of his daughter. The physicians and attendants left immediately."

While this may be overdrawn, we usually see general alarm, quarantine of suspects, house inspection by ignorant, brutal and sometimes dishonest and dissipated officials. Even in intelligent localities sometimes men of standing guard their neighborhood with shotgun cordons, giving an example of fear and lawlessness worse than pestilence. Wrecking of houses set apart for smallpox patients is a common occurrence. It is not many years since two expressmen were quarantined in Brooklyn because the commissioner of health thought they might become exposed to smallpox or carry it in the course of their business. The disease was prevalent and they refused to be vaccinated. Habeas corpus proceedings were necessary to release the victims from the "unwarranted and extraordinary" situation, to use the words of the judge in the final trial.[3]

Great expenditure of money is an essential part of the present method. Few places have special hospitals, and still fewer permit smallpox patients to enter general hospitals. Unfit locations, naturally and accurately termed pest-houses, must be obtained at exorbitant cost; special physicians, attendants, guards, and supplies raise the expense still higher.

But perhaps the greatest evil of all is the fact that at such times most of the vaccination has been done, by unfit persons often, on an alarmed and uninstructed public, with inferior and often wretchedly faulty virus. No wonder that some health officers, like the one in Cleveland, in 1902, stop vaccination in sheer terror at its abuses, and, depending upon the baseless hope of disinfection and quarantine without vaccination, invite still worse disasters. Such experiences form the most effective weapon in the scanty arsenal of the antivaccinationist, like an old-fashioned blunderbuss, useful only against a rash but undisciplined foe.

But these experiences we cannot escape so long as we adhere to our present method, or lack of method, of managing smallpox and vaccination, and so long as human nature remains as it is. Periods of relative freedom will occur, lasting for a few years, and then, as soon as a sufficiently large unvaccinated population collects, chance infection will start up epidemics, as it has hundreds of times in the past. If there was no other way, if we were as helpless now as we were 200 years ago, or as we are now against cyclones and earthquakes in their peculiar areas, the condition would be bad enough. As it is, we have the lesson of an immense experiment, carried on long enough and on a scale large enough to furnish conclusions of definite value.

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[3] In re Smith et al., N. Y., 146, p. 68, 1895.

This experiment has been made in Germany. The population there is not as large as our own, but large enough for a just comparison. (U. S., 1900, 76,303,387; Germany, 56,006,254.) The German people have no keener interest in private hygiene than our own, and public sanitation, in which they excel us, plays a minor part in the prevention of smallpox. The country is surrounded on three sides by neighbors with whom it carries on active intercourse, and all of these other peoples, up to the present time, have been relatively less protected by vaccination. Smallpox has been fairly prevalent in Austria, Russia, and France, less so in Holland, Belgium, and Switzerland, but always more than in Germany. For example, Warsaw in 1900 had more than 350 deaths from smallpox; Austria 999 cases and 143 deaths in the same year. The German vital statistics are kept with great care. It is a lesson in public service to read one of the annual reports of the Imperial Health Office.[4]

Smallpox Cases and Deaths in Germany.

Year.	Cases.	Deaths.
1900	390	48
1901	375	54
1902	114	16
1903	172	19

The tables show that in the same years I have quoted for the United States, Germany had only a fraction of our smallpox morbidity and mortality. The experiment further shows that this difference is not due to lucky chance. Similar favorable conditions have prevailed for the length of a human generation. Before that, from a short time after Jenner's discovery was published, many German states were distinguished alike by widespread and careful vaccination and favorable smallpox statistics; but this was not as perfect as many antivaccination writers assume. In 1870–71 all Europe suffered seriously, but, as an unusual feature of war times, the German army had a smaller proportion of cases, not only than the opposing army, but also less than the civil population at home, of corresponding ages. The only explanation for this was the more perfect vaccination of the military. Soon after the formation of the Empire, in 1874, systematic vaccination and revaccination were required by a carefully prepared law, and since then it has had a freedom from smallpox such as no other country in Europe or America can boast. Moreover, the few cases that do occur can be traced in large part, one-fourth in some years, to persons who had entered the country from a less protected land—railroad laborers from Italy, brickmakers from Hungary, farm hands from Russia. Some can even be traced to our own country, as in 1901, when several cases were due to infection from Utah, carried by delegates to a

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[4] Taken from the Med.-Stat. Mittheilung a. d. Kais. Gesundheitsamte.

Mormon Congress! Another large proportion is in infants not yet vaccinated. Vaccination and revaccination are the only possible causes of the difference, for shotgun quarantines are unknown and isolation hospitals hardly ever used for smallpox patients. Some are treated in their homes; some in general hospitals, even in wards with other patients. The disease rarely spreads beyond the first case. No one in Germany thinks the methods perfect. They are improved from time to time in various ways. Doubtless the future may show a better way of preventing smallpox than the present German method. Hitherto it has not been found. Some other countries have recently imitated it, with unessential modifications, notably France, Japan, Spain, Portugal, Argentine; even Turkey is making an effort, and Brazil would have adopted it but for a close relation between the vaccination project and an unlucky political party.

The two great countries that speak Jenner's language are among the last to adopt a practical and efficient system and also among those most devastated by smallpox.

In the United States the conditions are peculiar and extremely complicated. Many imagine that, owing to the high degree of intelligence of the public, voluntary vaccination is perfectly carried out. If this were so some other explanation than the one I have given for the existence of smallpox epidemics would be necessary. So careful a student of the subject as Voigt, in a recent report, quotes the *Lancet*[5] to the effect that 90 per cent. of our population is vaccinated and about half revaccinated. I cannot tell how the *Lancet* correspondent got his information. Early in the epidemic I made an examination of several hundred adults from all parts of Michigan and a few from adjacent States, where the vaccination laws are about as good and about as well enforced as the average, and I found only 60 per cent. vaccinated, and of these the majority had scars that were not at all satisfactory. Even public institutions, such as insane asylums, contained many unvaccinated people. In one of the best, out of 50 inmates who got smallpox in a short time, in 1901, only 17 had been vaccinated, "none recently, and with some the scar was barely visible."[6] Since then there has been much smallpox all over the State and a great deal of wholesale vaccinating, but in 1905 I found one-third of adult hospital patients wholly unvaccinated and almost none with good scars.

Voluntary vaccination can never be depended upon to prevent smallpox epidemics, nor should it be, for it is not done only for the protection of the person vaccinated, but for all. Moreover, the protection of the individual may be imperfect, and the population in general gets good results only when a large proportion of all are most thoroughly protected. It is true that some cities, such as New York and Chicago, have been pretty thoroughly vaccinated for some years past, but the conditions over the country in general are more important, and even lessen the value of the thorough vaccination in a few places.

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[5] 1905, ii, 1142.

[6] An Epidemic of Smallpox at the Michigan Asylum, etc., Arthur MacGugan. Medical News December 14, 1901.

The error regarding the extent of efficient vaccination has caused one of the most serious fallacies of antivaccination writers. They do not seem to appreciate the obvious fact that imperfect protection is the result of inefficient vaccination, and they make much of the sickness and death among those supposed to be but not really vaccinated.

There is a prevalent idea, even among many medical men, that vaccination can never be regulated by law in the United States. As a matter of fact, it not only can be, but in many States is the subject of statutes, as I shall show in detail later. The chief cause of misunderstanding comes from ignorance of the fact that vaccination, like other questions of "public safety, health, and morals," comes under police regulations, under the jurisdiction of the separate States; but there is no real obstacle to efficient and even to uniform statutes in every State as soon as people want them. Perhaps some way will be found in time to place so important a matter as vaccination under federal control, either by an extension of the principles of the interstate commerce law or an expansion of the public health service. Central regulation would undoubtedly be an advantage. Taking, however, only the means at present available, we find that States can enact satisfactory vaccination laws if they desire to. "In a free country, where the government is by the people through their chosen representatives, practical legislation admits of no other standard of action (except that based upon common belief), for what the people believe is for the common welfare must be accepted as tending to promote the common welfare, whether it does so in fact or not."[7] The most important decision up to the present is that handed down by the Supreme Court of the United States, in Jacobson *vs.* Mass.,[8] affirming the decision of the Supreme Court of Massachusetts.

The law contested was a statute authorizing boards of health to enforce vaccination and revaccination of all inhabitants, and imposing a penalty upon everyone over twenty-one years of age, and not under guardianship, who refused or neglected to comply. In the same statute an exception is made in favor of "children who present a certificate, signed by a registered physician, certifying that they are unfit subjects." The discrimination alleged as to this exception was brushed aside as unessential. The defendant claimed that the statute was in derogation of the rights secured to him by the preamble of the Constitution and the Fourteenth amendment. The court made short work of both claims. "Personal liberty," it said, "is not invaded when the State subjects (one) to fine and imprisonment for neglecting or refusing to submit to vaccination." It denied "that a competent vaccination law is unreasonable, arbitrary, and oppressive, and therefore hostile to the inherent right of every freeman to care for his own body and health in such a way as to him seems best. But the liberty secured by the Constitution of the United States to every person within its jurisdiction does not import an absolute right in each person to be, at all times and in all circumstances, wholly freed from restraints. There are manifold restraints to which every person is necessarily subject for the common good. On any other basis organized society could not exist with safety to its members. Society based on the rule that each one is a law unto himself would soon be confronted with disorder and anarchy."

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[7] Matter of Viemeister, 179 N. Y. 235, 72 N. E. 97. Am. State Rep., vol. 103, p. 863.

[8] 26 Sup. Ct. Rep., 358.

No objection should be possible against the principles expressed in the above, and in any event the decision is likely to influence strongly all future questions touching the constitutionality of vaccination laws.

An acute legal writer[9] has recently drawn some far-reaching conclusions from decisions in cases involving vaccination laws that deserve careful attention from physicians. He shows that a uniform course of decisions establish the fact that it is a valid exercise of the power of police regulations to require vaccination as a condition precedent to the right to attend school. "The natural question arises: Have these decisions paved the way for future declarations in favor of the right of the Legislature to order compulsory vaccination of all persons—adults as well as children? Any requirement of law," continues the editor, "to be sustained as a valid exercise of police power must be a reasonable requirement." The courts have been guarded in expressing an opinion as to the preventive power of vaccination. Their decisions are usually based upon "common opinion," "the present state of medical knowledge," or "common belief." "The efficacy of vaccination is regarded by the courts not as an established fact." "But if a mere popular belief is sufficient to uphold a police regulation for one purpose, why not for another? If vaccination is a reasonable requirement as a condition precedent to the right to attend school, why is it not also a reasonable requirement as a condition precedent to the right to live in a community? In other words, if the legislature can say that no child can attend school unless he has been vaccinated, why can it not also say that no person shall reside within certain corporate limits unless he shall have been vaccinated?" This view, in fact, was upheld in a case in Georgia.[10] As regards school attendance, this is not merely a right or a privilege. In many

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[9] Law Notes, December, 1904, p. 405.

[10] Morris *vs.* Columbus, 102 Ga. 792.

States it is compulsory. So it was decided in Matter of Walters,[11] that "inasmuch as attendance at school is compulsory and vaccination requisite as a right to attend school, vaccination is compulsory." As the editorial in question also says, "Courts under certain circumstances must uphold compulsory vaccination, even though forcible vaccination of a person against his will may be technically an assault."

In order properly to understand the present status and especially the imperfections of our vaccination laws, it is necessary to consider some of the enactments in detail.

Many States have no statutes regarding vaccination. In 1905 about twenty were in this condition, including some, *e. g.*, Illinois and Indiana, of great importance in population and geographical position. In such States, as in others, unless there is a statute to the contrary, the health authorities may make their own regulations, within certain limits, with or without special delegation of power for that purpose.

In Arizona (1903) general vaccination is obligatory, but the details to be observed are not prescribed. In Kansas,[12] "In case of smallpox a general and thorough vaccination shall be recommended and insisted upon." In Kentucky[13] all must be vaccinated, adults and minors; infants within twelve months of birth; persons coming in from other States, "to abide or become citizens," within six months, if not previously vaccinated. In Maine[14] vaccination is obligatory on all inhabitants over two years of age. In Maryland[15] all infants must be vaccinated within twelve months of birth. In Massachusetts[16] vaccination must be carried out in all persons under two years of age, unless excused on certificate of a physician by reason of unfit physical condition. In Mississippi a compulsory vaccination law was enacted in 1900, and the law amended in 1902, but the details are left to the local authorities. In West Virginia (1905) compulsory vaccination may be ordered upon petition of 100 voters of a county, in case of smallpox epidemic.

Revaccination is rarely mentioned, though often ordered indirectly. In some States general vaccination may be carried out in case of threatened danger from smallpox—Georgia, Massachusetts, New Hampshire, North Carolina, Oregon, Tennessee, Vermont, etc.

In Connecticut and Maine employees in paper factories are required to be vaccinated. Employers (in Connecticut) are obliged to "pay to any town all expenses caused it by the sickness of such person with smallpox contracted while so exposed." In Maine revaccination must be repeated in two years and all employees in paper mills must be examined twice a year with reference to the thoroughness of the vaccination.

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[11] New York, 84 Hun. 457.

[12] Gen. Stat. 1905, Sec. 3438.

[13] Carroll Stat. 1903, C. 119.

[14] Rev. Stat. 1903, Chap. 18.

[15] Public General Laws, 1904, p. 1211.

[16] Rev. Laws, 1901, p. 682.

In Kentucky and North Carolina inmates of charitable institutions and the penitentiary must be vaccinated. In Massachusetts they may be at the option of the board of health, as may also operatives of manufacturing establishments.

In many States vaccination is a prerequisite to admission to the public schools. This is sometimes optional with local health boards, as in Connecticut, Georgia, Maine, New Jersey, Ohio, Oregon, West Virginia, and Wisconsin. In Wisconsin it has been decided that vaccination cannot be required as a condition precedent to the right to attend school.[17]

In some States the requirement is mandatory: the District of Columbia, Maryland, Massachusetts (except when excused by reason of unfitness), New Hampshire, New Mexico, New York, Pennsylvania, Rhode Island, South Carolina, and Virginia. In the Indiana school law there is a clause: "No child in good health and free from infection shall be excluded from the schools of Indiana, any law to the contrary notwithstanding." In Illinois[18] it has been decided that directors have no power to compel vaccination in the absence of smallpox. In Connecticut the existence of smallpox is not essential. In Minnesota[19] compulsory vaccination of school children is prohibited, except in case of epidemic, in children not excused by certificate as unfit. In Louisiana (1897) compulsory vaccination is prohibited. In South Dakota[20] physical force may not be used in vaccinating. In Utah a law to prevent compulsory vaccination was passed over the governor's veto in 1901. Teachers as well as pupils are required to be vaccinated in Virginia, Wisconsin, and also in New York City. Pupils of private schools are mentioned only in the laws of South Carolina.

The Territory of Hawaii[21] has a better law than any American State, but it does not go as far as is desirable. It includes such important desiderata as: A special vaccinator in each taxation division (other physicians may vaccinate); at least three convenient places in each school district for the performance of vaccinating; the operator must give notice of the times set for vaccinating and for inspection of results; these times must be at least once in every six months, oftener if required by the board of health; records must be kept and reports made; every child must be taken to the officer within six months of its birth, "or at the earliest opportunity after;" the child must be taken upon the eighth day after the operation so that the officer may ascertain by inspection the result of the operation; the result is to be noted in a book, and in case of successful vaccination a certificate given; if the child is unfit, the vaccinator may postpone the operation and notify the parent or person in charge of the "child to reproduce the same for vaccination at a future time;" there is a fine of five dollars for failure to observe the statute: "No child shall be admitted to any public or private school without producing a certificate of vaccination or showing marks of successful vaccination," under penalty of five dollars on the part of the teacher. Only bovine virus is to be used.

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[17] State ex rel. Adams *vs.* Burdge and others, 95 Wis. 390.

[18] Potts *vs.* Breen, 167 Ill., May, 1897.

[19] 1903, Cap. 299, p. 530.

[20] 1903, p. 297, Cap. 223, Sec. 1.

[21] Rev. Laws, 1905.

From the summary I have given it is obvious that there are very great differences in the scope of the laws regarding vaccination in different parts of the Union. This is not due to failure of sufficient constitutional basis for sound laws; nor is it due to public opposition to sound laws. Objections are made not so much to the statutes as to the manner in which they are enforced, or without any definite reason. Kentucky has one of the best laws, but there is no more opposition to it than there is to many weaker laws. Some of the statutes are so worded as to rouse latent or open antagonism even on the part of those who should enforce them. For example, in Maryland (1904) it is permitted to use human virus, "if practicable not more than four removes from the cow," a practice abandoned by all careful vaccinators. The fact that there is a penalty of one hundred to five hundred dollars for using defective virus does not relieve the anachronism of the earlier section. It is the duty (Sec.43) "of every practising physician . . . . to vaccinate all children in the circle of his practice which may be presented to him for vaccination, within one year, if such child shall be in a proper condition for such service;" and "all other persons not previously effectually vaccinated who shall request such service from him. Any physician neglecting or refusing so to do shall, on conviction thereof, forfeit and pay for every offense a penalty of five dollars." The advantages to be gained from the law are certainly not enough to warrant such demands.

In West Virginia, in the law of 1899, it was provided that certain agents were to send "genuine vaccine matter" and "directions how to use it," by mail or otherwise to every citizen of the State who applied for it, showing an enviable absence of the anti-movement, and a gross ignorance of the operation and its objects. In 1905 the law was amended, and "local option" vaccination and quarantine permitted in case of smallpox epidemic. "Competent physicians" can be appointed as public vaccinators, but in no case can they receive more than twenty-five cents for a successful vaccination, and no compensation for an unsuccessful vaccination. As "such physicians" have to provide their own virus, they can hardly fall under that part of the antivaccination complaint which alleges the medical profession upholds vaccination on account of the lucrative fees that the operation provides. As the State does not furnish any guarantee of the quality of the virus in the market, the injustice of the law is obvious.

A careful perusal of the statutes gives the impression they were devised without reference to the best methods available. The legal part is usually correct, but the medical part seems to have been overlooked almost uniformly. A surprising lack of care is especially striking in the South Dakota statute of 1903:

"Sec. 1. It shall be unlawful for any board, physician, or person to compel another by the use of physical force to submit to the operation of vaccination with *smallpox* or other virus.

"Sec. 2. No person shall prevent a child of school age, who furnishes a physician's certificate of successful vaccination with *smallpox virus* within five years, from attending school."

It would be tedious and not profitable to discuss all the weak points in the various laws, and I shall criticise only the general bearings and important special features.

The laws are far too vague and uncertain to give such protection as is needed. It may be thought, and probably was supposed in framing statutes, that the best results could be obtained by making only general laws and leaving details to the officials who have to interpret and enforce them; but it is no more possible in this way to secure good results with vaccination than with other duties of society. We might just as well leave the protection of water supplies to public benevolence, as in fact we often have, with uniformly bad results. What we need is the widest possible application of the most efficient vaccination. Under any arrangement numerous officials are necessary for this. The best results so far have been obtained in certain cities where energetic and capable physicians have devoted themselves to the work. But men of that kind cannot be secured everywhere or always. Some are ignorant; some lukewarm; some opposed to the views or personalities of their superior officers. They may be hampered by red-tape or by lack of support, either financial, moral, or physically, from their superiors. So far as possible, then, accidental causes of failures should be reduced to the minimum by more explicit laws and more care in selecting officials.

The necessary thoroughness cannot be attained without accurate registers, which shall show the true condition of all persons with regard to perfection of vaccination and revaccination. We already have learned the advantages of lists of voters and of school children. It would add much to public order, and much advance the efficiency of vaccination and other sanitary details, to enlarge these lists and to enter various data regarding health and disease.

The method of combining vaccination and school attendance is very irrational, and yet in many States it is the main dependence. If vaccination is not done until the child begins school, as the natural tendency is at present, we leave the most highly predisposed part of the population—children under five or six years of age—wholly unprotected. A considerable part of the population may go unprotected even longer, because the children attend parochial or private schools, which in most States do not come under the statute.

The responsibility for the performance and even sometimes the success of vaccination is usually left to the teachers, who have not the proper training, and in most cases have not time to make the necessary examinations.

The laws sometimes require the impossible—*e. g.*, "successful vaccination of all pupils," Maryland, Pennsylvania; "successful or proper," Virginia; "successful" or "within one year," New Mexico; "proper," Rhode Island.) I do not mean that all people may not be successfully vaccinated. We all know that many vaccinations do fail. Even in Germany the primary success is not better usually than 85 per cent., varying from about 60 to less than 100 per cent. Not a few fail up to three attempts. In a recent news item,[22] it is stated that in the autumn of 1906 in Los Angeles, California, "more than 1000 primary vaccinations failed." "As the result of hot weather and exposure of the virus to high temperature *en route*," the account goes on to say, but the same exposure *en route* occurs all over the country, overheating in railroad cars being one of the stock excuses of the makers for weak virus.

The result is that a low standard of success is tacitly accepted everywhere, giving brief immunity or none at all. It is encouraged by assertions of makers of vaccine virus, by laws such as the one in West Virginia, and by the indifference to or lack of a careful noting of the results. In many places the patient is never seen by the vaccinator after the operation. And from the standpoint of education it is well that this is so. Notwithstanding all the current criticism of the public schools, in our present stage of development they are better than none at all. But if both education and vaccination are good, how unfortunate that they should be made to appear antagonistic, as they are at present in the public mind!

As a matter of fact, our experiment makes it plain that so theoretically perfect a requirement as we have in our school and health laws is not at all necessary. In Germany most children are vaccinated before they are two years old, and again at twelve. But there are many exceptions at both periods. From illness of various kinds many children are excused; in some, vaccination fails, but a certificate permitting school attendance is given (as it is in some few States in America). A few children remain unvaccinated illegally, or are imperfectly vaccinated and false certificates given. It is easy to see that these failures may in time become a source of great danger. Up to the present they have not been so great a source of danger as our stricter but impossible requirement. It is interesting to see that in Germany the question of compulsory vaccination has been decided in diametrically opposite ways by courts. Some fathers have paid their fines every year, on account of the non-vaccination of their children. Another obtained a decision showing that compulsory vaccination could not be made a condition precedent to attendence at school.[23]

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[22] Medical Record, October 27, 1906, p. 660.

Quite as faulty in its results is the law providing for vaccination in time of epidemic of smallpox. Even when the time is not left, as in West Virginia, to the good-will of the voters, the method has several weak points. Vaccination under the circumstances is too late for many people, as it takes time for the diagnosis to be made and the work of vaccination to be set in motion before a number of people are involved. Many people escape the operation, very often those who need it most. In emergency vaccination there is no consistent attempt at controlling or revising the result; many people who imagine they are well protected with or without scars, acquire the disease sooner or later afterward.

As epidemics often occur in different parts of the country about the same time, there is an excessive demand for vaccination, and inferior vaccine is put on the market, giving imperfect protection and more frequently causing septic infection. Sometimes there are other epidemic diseases at the same time that make operations dangerous—erysipelas, even tetanus. Owing to the haste required to vaccinate large numbers in a short time, those who know better hurry too much, many others who do not know assist in the work—physicians, servants or children, drivers, office-boys, or policemen. Many of the sore arms occur under these circumstances, either from careless operations, bad vaccine, or from operating on people who should have been excused. In the United States Supreme Court case of Jacobson *vs.* Massachusetts it was given out as impossible for unfit people to be vaccinated. It did not seem necessary to have a definite statement in the statute. As a matter of fact, we all know that many unfit people have been vaccinated, sometimes by force, with great brutality, and often with serious or even fatal results. There is no consolation for the parents in such cases to know they can bring suit for assault.

The omission of vaccination in such cases would not be nearly so dangerous as another method, legalized in one of the States, and practised in many—that of not vaccinating people living in sparsely settled regions. Yet these people very often have smallpox affecting patient after patient, often not seen by a physician, but occasionally moving to another place. Several patients have been admitted to the isolation hospital in Ann Arbor, having travelled many miles, sometimes hundreds, with smallpox. How many people exposed on trains, in stations, or in hotels have taken smallpox from such patients I cannot, of course, tell. From one sparsely settled district a woman travelled some distance with the eruptive stage well advanced, attended a wedding, and infected many people who reached almost as many towns before they knew what risks they had run. At least six are supposed to have taken the disease from her. The experience of the hospital follows the universal rule—no student, physician, nurse, or patient has taken the disease from any of the smallpox patients. All of these are vaccinated as carefully as possible. One case only occurred from infection in the hospital, an orderly who was wrongly supposed to have been recently vaccinated. He was exposed only for a few minutes, while helping to carry a patient on a stretcher. The husband, who carried the other end, and was kept in the smallpox hospital, was vaccinated at once and did not take the disease.

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[23] Med.-Stat. Mittheilung a. d. Kais. Gesundheitsamte, Bd. viii, p. 61.

All the faults mentioned depend upon the method of carrying out vaccination, and could be avoided by systematic vaccination and revaccination. Others, just as serious, exist in connection with the material used. Many of the laws were passed before there was any control of vaccine virus, although there was plenty of evidence that it was often faulty and that there was no way for the practising physician to recognize the good or the bad. The high bacterial content, the poor specific power, the insanitary condition of some factories were all known. Fortunately there has been a great improvement in the material since the United States Public Health and Marine Hospital Service took charge of the inspection, but there is room for still further improvement in the potency of the material. From the fact that some of the factories had to be closed on account of their bad condition, and from the misstatements of the makers, it is evident that the control should not be relaxed, but should become closer. At the same time the freshness of the virus should be more closely regulated and the factories distributed with a view to better service.

Perhaps more than anything else we need educated and trained vaccinators, and these should have positions that would enable them to carry out their functions as they should be. To make vaccination compulsory without providing educated vaccinators is as irrational as it would be to make a law requiring railroad companies to take untrained men as train dispatchers or locomotive drivers. I am not aware that this feature of the laws has ever been tested in a court. It cannot be emphasized too often that vaccination to be most successful must be a public matter, not dependent upon the good-will of the individual or the peculiarities of the private physician. In one city often visited by smallpox, I am informed that the public vaccinator refuses to vaccinate those exposed in smallpox houses, on account of a theory he holds regarding the danger of such an operation. In Germany a relatively large proportion of bad results come from the few persons vaccinated privately.

Perhaps a more efficient system of vaccination would cost more than the present method with its high smallpox morbidity. I know of no trustworthy data. For England, Mrs. Garrett Anderson has estimated that thorough vaccination would cost less than the present system. We have not the isolation hospitals that England keeps up at great expense, but I believe that our haphazard system is proportionately more expensive. The German establishment is expensive, because skilled men and the best possible equipment are obtained. But practically nothing is spent for special hospitals, and very little for quarantine or the indirect expenses we have to meet. Moreover the investigations that come out of the government laboratories of Germany, and the same may be said of those in France and England, more than pay for the best that can be obtained, by adding to our knowledge of vaccine and its effects, and by perfecting the practical details of raising, keeping, and using lymph.

In conclusion I would make the following summary of the vaccination situation:

1. Our present method, varying much in different States and different parts of the same States, makes certain a high ratio of smallpox cases, with an unduly low protection to the individual.
2. There is nothing in the fundamental law of the land to prevent the passage of safe and efficient vaccination laws.
3. Vaccination laws should aim at a widespread protection, by vaccination and revaccination.
4. Compulsion should not be necessary, and should by no means take the place of safe and certain vaccination. Compulsion should not be practised in case of doubt as to the fitness of the subject by his physical condition or surroundings.
5. The operation should be a matter of permanent record, and a certificate from an authorized official should be proof of the vaccination of each individual.
6. The operators must be trained for their work, familiar with the vaccination laws, and bound to follow them.
7. The manufacture of vaccine must be controlled by competent experts; institutes for furnishing vaccine should be so placed as to make easier the distribution of virus.
8. The operation should be done at fixed times of year, when epidemic diseases are not most prevalent, in places appointed and equipped for the purpose; the subject should be examined after the operation at a time fixed by the regulations, or at once on suspicion of complication. Private vaccination should be permitted only under special conditions and the proper details should be exacted, with revision of the result by a competent health officer.

THE CLINICAL MANIFESTATION AND TREATMENT OF SOME FORMS OF ACUTE CARDIAC DILATATION.

By Beverley Robinson, M.D.,
of New York.

Cases of chronically dilated heart, as a result of chronic endocarditis, are very frequently met with in practice. We also encounter them in persons, particularly men, who have well-defined signs of renal disease, or of general arteriofibrosis. Among the manual laboring class, strictly speaking, we find them often as a result of arduous and continuous work; or when frequent heavy strain has occurred, large, dilated hearts are not uncommon. Of course, with an antecedent history of syphilis, or chronic alcoholism, even though the previous life-work has not been unusually trying to physical strength, the dilated heart is likely to become manifest, and that, too, at a somewhat early date, because degeneration of muscular tissue, as a result of diseased arteries and insufficient cardiac nutrition, is without doubt a notable and important factor in producing it. Again, many acute febrile diseases, especially those of infectious nature, unless their convalescent period is most carefully and a long while watched and cared for, are followed, sooner or later, by cardiac dilatation—which has come on slowly and insidiously, perhaps, but when once developed is of bad augury for the future, since it can rarely be wholly cured and only occasionally ameliorated to such an extent that life is relatively free from painful or distressing symptoms.

But my purpose in this paper is to study, not these instances with which we are all familiar, but rather those to which attention has certainly been directed by many observers, but to which still belong, as I believe, a few notes, based on observation and clinical experience, that may add something of more than passing interest and importance.

I shall first give thought and attention to the cardiac dilatation which shows itself during the course of some acute infectious diseases, such as rheumatism, pneumonia, diphtheria, or influenza. I shall then speak of acute cardiac dilatation of nervous origin, due primarily many times to the wear and tear of an anxious and working life, but in which the strain falls far more directly upon the brain and nervous system generally than upon the muscular make-up of the body. Finally, I shall speak of rapid or sudden cardiac dilatation which comes to those whose excesses in eating, or drinking, or the use of tobacco, are notable and long continued, and who are unable to bear even temporary physical strain without experiencing painful, threatening, or alarming effects which become manifest very soon or immediately.

CARDIAC DILATATION IN ACUTE INFECTIOUS DISEASES.

Acute rheumatic endocarditis in children results fatally, not by reason of the endocarditis *per se*, but because of the cardiac dilatation which follows it and is "due to a toxic action of the rheumatic poison on the cardiac muscle" (Lees). Certainly, there is much to justify this conviction if we consider the revelations of many autopsies. In some cases, no doubt, the pronounced chronic valvular disease will sufficiently explain the cardiac enlargement; but in not a few cases the endocardial changes are relatively so slight that we are not satisfied they could have been the sole and sufficient cause of the cardiac dilatation. In view of Poynton and Paine's researches, the diplococci found in the heart wall, or their toxins, are probably the really efficient factors in producing it. This dilatation "tends to diminish as the rheumatic symptoms subside, and it may increase again if the rheumatism relapses."

In many cases of rheumatism, acute dilatation of the heart is far more important in its immediate and remote results than any pericardial rub, or endocardial murmur. Indeed, it seems as though it were primarily, to a considerable degree, the direct result of the action of the rheumatic poison on the cardiac muscle itself. Its remedial treatment consists largely in the proper use of antirheumatic remedies, that is, internally, salicin or the salicylates, and sodium bicarbonate, and, locally, bloodletting with leeches or venesection, followed by applications of the ice-bag. In this way the heart can often be notably reduced in size, or, indeed, completely restored to its normal size, and also to its normal strength. If, however, the treatment is lessened too much, or stopped too soon, a relapse to rheumatic symptoms and particularly to cardiac dilatation is almost sure to occur. This treatment, according to Lees,[1] should be large doses of salicylates, combined with double the quantity of sodium bicarbonate to prevent salicylic acid poisoning and the "air hunger" so characteristic of it. In rheumatic children he finds "that after a short time 300 grams of sodium salicylate and 600 grams of sodium bicarbonate (in ten doses) are generally well borne, that they rarely cause vomiting, and that no depression follows." In many cases "the large doses have been continued for two or three weeks."

These facts, if duly corroborated by others, appear to me of the highest value. Locally, whenever there is engorgement or dilatation of the right ventricle, leeches should be applied; subsequently the ice-bag should be used more or less continuously. The ice-bag is of service in subduing the inflammation present and in stimulating and quieting the heart. If these methods were adopted generally they would very greatly diminish the subsequent evil effects of acute cardiac dilatation in rheumatism and probably prevent a great amount of cardiac misery. No one who reads Dr. Lees' recent work carefully can fail to be forcibly impressed with the care and accuracy of his clinical observations. It is evident how important such findings are; it is also evident how valuable the methods of treatment, particularized and insisted upon, may later prove to be.

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[1] Acute Visceral Inflammations, London, 1904, p. 296.

In *pneumonia*, especially in the cases that are serious or imminent from the point of view of the profound general toxemia or from the extent of the pulmonary obstruction, dilatation of the heart in acute form is likely to be the subject of our greatest, most immediate anxiety. In the severe toxemic cases the whole heart is involved, and no doubt the pneumococcus, or its toxin, affects the muscle in its entirety and produces relaxation, loss of tone, even degeneration, hence rapid or even sudden loss of strength in cardiac systole, and a weak, failing, rapid, or intermittent pulse. Even in these instances, and until death is very near and the respiratory centres are directly attacked by the systemic poisoning, the pulse has an appreciable volume, although it may be easily compressed and almost obliterated. The picture is very different when the right heart dilates and fails from its inability to overcome the ever-present and perhaps increased pulmonary obstruction. In these cases, it is true, the pulse is very small, and even apparently weak. But it is not because the left ventricle has lost power or is flabby and degenerated. Rather it is that the ventricle does not receive blood enough from the lungs to contract upon sufficiently and to cause an appreciable pulse wave. Under these circumstances, if we percuss the heart area we find notable increase of dulness toward the right, and sometimes in the intercostal spaces on this side, notably the third and fourth, we may detect a pulsation synchronous with the cardiac beats. Moreover, there may be marked epigastric pulsation and a considerable distention of the large veins of the neck. At this stage the pulmonic second sound, which previously had been notably accentuated, owing to increased obstructive conditions in the lungs and too rapid and forcible arterial recoil under the dependence of a struggling although powerfully acting right ventricle, has rapidly lost accentuation and become so weak as to be scarcely audible. If with this loss of pulmonic second sound, and even though the pulse remain very small, there is no increase of or very slight dyspnœa, it may be that pulmonary obstruction is diminishing, and the prognosis of the case is brighter and more hopeful. If, on the contrary, the dyspnœa is augmented and cyanosis more marked the outlook becomes, for the while at least, graver.

In such cases A. H. Smith,[2] who many years ago described, very graphically, the conditions alluded to, would insist upon the wisdom of making immediate and sufficient use of the vasodilators, and especially of the nitrites. The major and urgent indication is, so to speak, to bleed from the veins into the arteries. The former are blocked up and their circulation intensely impeded by an overflux of blood; the latter receive very little blood, and the left heart is absolutely prevented from exercising its great power by reason of insufficiency of fluid in its cavity to permit of efficient action. If, under these circumstances, drop doses of nitroglycerin solution are given every fifteen minutes or every half-hour, we may at times, and very soon, see a very great and happy change for the better. The pulse increases considerably in volume, the veins in the neck become less distended, the dilatation of the right heart in part disappears, the right ventricular systole is more effective, and the pulmonic second sound is again audible.

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[2] Amer. Jour. Med. Sci., 1890, p. 397, and Med News, December 16, 1899.

Dr. Smith claims that in such cases the nitrites are of greater value and freer from objections than the time-honored venesection. In this I am not inclined to agree with him. On the contrary, I hold that the preliminary bloodletting, at least in moderate quantity, is essential, as a rule. I prefer, however, the application of leeches or wet cups locally to the use of the lancet: (1) Because I believe either of these methods causes less nervous upset than venesection; and (2) because despite much careful reasoning based on physiology of the circulation which declares the same amount of blood taken from a vein to be equally as beneficial as a similar amount taken locally, I still believe the contrary. I am satisfied that local bloodletting is more effective and sooner relieves symptoms of distress and oppression than venesection—the amounts of blood abstracted bring equal in the two cases.[3] I believe that the reason for this may be discovered, not in the amount of blood taken from the patient, but in the reflex started locally in the right ventricle that is transmitted to the respiratory centre, and from there becomes additionally stimulating to the heart. It is also true that the vasomotor nerves are called into action through this reflex, and afford relief through dilatation of the peripheral arteries (Lees). In this condition of pulmonary obstruction and dilated right heart we should beware of the use of digitalis in preference to the vasodilators. Rarely, if ever, should we expect benefit from digitalis; on the contrary, we find rather increase of cyanosis and distention of the veins. Keep the color florid with the use of nitroglycerin, oxygen, alcohol, and suitable diet, and, as Smith writes, we shall not be ashamed of the teachings of the sphygmograph. It is far better to have a full, gaseous pulse than a small, thready one. Smith has never yet seen a small, thready pulse become fuller and slower from the use of digitalis. It is true, however, and important to add that after the distention of the right heart has been relieved by leeches or venesection, digitalis may become very serviceable in giving strength to cardiac beats when the pulse is weak, rapid, and intermittent. Digitalis is also sometimes useful, in my judgment, when the heart as a whole is affected and weakened by the toxemia of pneumonia—not merely its right auricle and ventricle because of the pulmonary obstruction.

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[3] In this opinion I regret to find that I am not in accord with Roland G. Curtin, who says: "There is, I think, very little, if any, benefit in local bleeding," Trans. Amer. Climat. Assoc., xxi, 178.

In *diphtheria*, especially in the marked toxic forms, it not infrequently happens that sudden or very rapid death takes place. This fatal termination may come during the first days of the attack. With other signs of intense poisoning the heart impulse may increase much in rapidity and feebleness. Together with these signs there may be increasing dyspnœa, pronounced pallor of the face, and cyanosis of the lips and fingers. The face itself may partake somewhat of a dusky hue, but it is the extreme pallor, ashy at times, which excites remark and absorbs attention. The combination of symptoms is of very bad augury. There may or may not be a considerable elevation of temperature (100° to 102°). The latter point is rarely reached and often we have only 0.5 to 1 degree of elevation. In times past I have seen many such cases, and, as a rule, prior to death there were evidences in several instances of a pneumonic process in one or both lungs which had already reached the stage of solidification. Death occurs, and the pneumonia is usually verified at the autopsy. If the heart is carefully examined, what is most marked is the great distention of the right cavities with wholly cruoric clots, like currant-jelly in appearance and consistence. In other cases, these clots are half or more cruoric, the remainder of the clot being fibrinous. A similar clot formation extends well into the pulmonary artery, and both the right auricle and ventricle are filled to repletion. The clots are formed during the agony, or one or more days preceding it. Whenever the clots are largely fibrinous and possibly give some evidence of having the fibrin deposited in distinct layers, but above all when the marks of the pulmonary cusps are distinctly impressed upon the arterial clots and all the cruor is pressed out of them, we may feel quite confident that these thrombi were formed some time (one or more days) previous to the fatal termination. In such cases the heart muscle, if carefully examined microscopically, will show very few changes. The striæ are sometimes less well-marked and the muscular fibers are somewhat granular. It is rare, however, to find distinct fatty globules anywhere in its structure. Formerly it was stated, the pneumogastric nerves show no discoverable pathological changes. Latterly, however, Councilman and others have found nerve changes, although Schamshin denies them. The degeneration of the heart muscle is admitted by many. It seems, however, to White and Smith that degeneration of the vagus was indicated only in the severest type of heart cases. In this connection they write, "Undoubtedly heart failure not infrequently results from paralysis of the cardiac nerves (or cardiac plexus), and quite independently, too, of any anatomical changes in the heart muscle."[4] Lennox Browne credits Vincent, of Paris, and P. Meyer with having found "widespread parenchymatous changes in the cardiac plexus in two patients dying of heart failure during convalescence from diphtheria, in which the heart muscle was unaffected."

Experimentally it has been shown that the healthy heart can rarely be overdistended, no matter how much its working capacity may be overtaxed. This is not true when several injurious factors are combined, nor is it true when a previous infection has existed and has surely altered the heart muscle.[5] Colbeck[6] attempts to show that all forms of dilatation indicate an absolute or relative loss of muscular tone of the organ. When in diphtheria the heart itself is somewhat dilated this dilatation affects the right cavities almost exclusively; the left auricle and ventricle are relatively empty and so far as one may determine are of normal size, and the muscular degeneration is less marked even than in the right heart. Formerly, I regarded such cases as being due not infrequently to paralysis of the heart, which came about through lack of government of the pneumogastrics, occasioned, doubtless, by changes in the nerve trunk itself which escaped our careful examinations. Today I believe it more satisfactory to make a diagnosis of acute dilatation of the heart and to attribute rapid or sudden death essentially to this condition. Of course, we have the complicating pneumonia and heart clot, but these might not have occurred, or indeed proved fatal, unless for the development of extreme and fatal heart weakness caused by the poison of diphtheria. I have seen death occur suddenly more than once in diphtheria. I have seen it in children not only, as I have said, during the acute stage of the disease, but after days had elapsed and the patient was approaching the true convalescent period. In the latter period the patient has died suddenly when there was scarcely a warning to tell us to be especially careful, except the fact that the patient had had diphtheria.

I have had one of my adult patients die suddenly when doing apparently well and sitting up in bed and playing cards with his wife. This patient had been examined by the late Austin Flint only a few hours before his sudden death, and his heart had been pronounced in good shape and no danger was anticipated. In more than one other instance I have seen very alarming conditions of heart failure. This goneness, faintness—with weak cardiac impulse and frequent, depressible, feeble, and irregular pulse—has returned twice or several times, during a period of a month or six weeks after the acute disease had terminated. With the heart weakness—the syncopal attacks, when the respiration was very shallow and slow—there was a certain degree of paralysis affecting the soft palate, or some of the muscles of the thorax, or extremities. In such cases, when I have percussed and auscultated the heart, I have been quite sure that the heart was distinctly dilated, inasmuch as percussion and auscultation gave every sign of this condition. Such a condition is unquestionably remediable, not only as far as the symptoms are immediately concerned, but also in regard to their future continuance. Ultimately the heart can be completely restored and be considered as good as ever. In other cases, especially those of adults, I doubt somewhat whether we are authorized to make unqualifiedly a favorable prognosis.

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[4] Welch and Schamberg, Acute Contagious Diseases, 1905, p. 640.

[5] Med. News, January 9, 1904, p. 68.

[6] Lancet, April 9, 1904.

At first, when attacks occur, complete bodily and mental rest is absolutely required. The patient should lie down; everything tight should be loosened or gotten rid of; smelling salts; hot-water bags to the precordium and the extremities, and five minims of tincture of strophanthus with ammonia and brandy by the mouth, or alone hypodermically, will usually bring about a rapid temporary improvement. But to secure complete cure of an acute cardiac dilatation of this origin requires months and years. Overfatigue should be strictly avoided for many months. Iron, quinine, and arsenic must be given regularly for a while to counteract the injurious effects of the secondary anemia. Strychnine, in very moderate doses, will help the heart for a time and prevent in a measure the return of syncopal tendencies. After all, however, it is country air, good nourishment, and complete rest of body and mind which will be found really most efficacious. Time[7] itself will help greatly to eliminate the remains of the toxemia from the economy; by successful war the protective forces of the body, little by little, get the ascendancy and the heart itself becomes once more normal in size and force. I have seen more than once the slight beading or small warty excrescences along the free margin of the mitral valves at autopsies in cases in which death occurred during the acute period of diphtheria. I have every reason to believe that these growths are completely resorbed later, as it is a rare circumstance to recognize clearly chronic endocarditis which we could fairly attribute to a previous attack of diphtheria.

"Pathological studies have shown that the murmurs are probably due largely to relative mitral insufficiency resulting from changes in the heart muscle or to changes in the innervation of the heart."[8] It is questionable in what number of cases acute dilatation and syncope occur. Frequently the symptoms of heart failure come on gradually; sudden death is uncommon if rational treatment is followed. It seems evident that acute dilatation of the heart is less frequent in diphtheria than in rheumatic fever. Nevertheless, in the few cases of very slow pulse there was cardiac dilatation, and in those of gallop rhythm and very frequent pulse "the heart is usually moderately dilated. In practically all of the fatal cases the heart was dilated."

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[7] H. B. Anderson very properly lays stress upon the length of time after a severe acute disease that the heart should be watched and all undue strain avoided, even though no cardiac signs point to the existence of myocardial changes. "After a severe illness probably a year will be required in favorable cases before the organ has regained its former strength." (Brit. Med. Jour., October 7, 1905, p. 845.)

[8] White and Smith, Heart Complications in Diphtheria, Boston Med. and Surg. Jour., October 20, 1904, p. 423,

Influenza is another frequent cause of rapid cardiac dilatation. Even in the early days of the disease it may occur. Not infrequently the only satisfactory explanation of the obvious great weakness of the patient is to be found in this condition. Not only is acute cardiac dilatation not infrequently met with in influenza during the first week of the disease, but subsequently, as we know, it is also encountered. The patients often get up fairly well from an acute attack, and then after a few days have a sudden relapse of weakness and prostration, and are unable to meet courageously and well the ordinary duties of daily life. When the heart is auscultated and especially when it is percussed, cardiac dilatation is sometimes clearly discovered and satisfactorily accounts for many of the patient's symptoms. Again, a patient who has had a severe attack of influenza may, after a relatively long convalescence, appear finally to be completely re-established in health. Without apparent or sufficient cause, and despite great watchfulness and care, or sometimes, it may be, following some evident and perhaps great imprudence or strain, cardiac inadequacy manifests itself and the patient feels limp, tired, incapable of exertion. Often every other organ has been examined carefully—lungs, liver, spleen, kidneys, stomach, bowels, etc., and everyone seems in fairly good shape, until the heart is very carefully considered. Not seldom the dulness to the right of the sternum is apparently about normal, and there may be no marked epigastric pulsation or venous distention in the neck; but the dulness has gone up to or beyond the vertical line passing through the left nipple, and with it the first sound of the heart is indistinct, muffled, weak, distant. The area of pulsation is feeble and diffuse. The point of greatest intensity of the cardiac systole is not readily made out, nor is the apex-beat either visible or always palpable. The pulmonic second sound has lost the accentuation, and is often extremely indistinct, and rarely almost inaudible. "These cases," writes E. G. Wood,[9] "I take to be examples of toxic degeneration of the myocardium, with resulting loss of vitality and acute dilatation." With this opinion I cordially agree.

It is in these instances, when all ordinary means have failed to re-establish the health, and when the heart action remains attenuated and feeble, with symptoms almost of collapse at times, that an ocean voyage is sometimes most beneficial. Nothing seems to replace the absolute rest, peace, quiet from all disturbing elements, to the overwrought professional or business man, which thus can be obtained and only thus. Of course, the ocean breezes, the complete change of diet, the daily agreeable pleasant diversions on shipboard, are of untold utility to some. We must, however, distinguish at times between patients who love the sea and patients who do not; between patients who are never sea-sick and those who, perhaps, are not acutely ill, but, on the other hand, who are never entirely well. The latter class of patients is certainly far from being as promising for the benefits of an ocean voyage as the former. To these patients, as a final resort, comes the advantages resulting from the Nauheim treatment abroad, or at home. I have regarded and continue to believe that some patients—many perhaps—properly selected, get great and enduring benefit from this treatment wisely ordered. I certainly believe that there are cases of cardiac dilatation following influenza, which show a disposition to recur and become very threatening, in which Nauheim treatment has been most beneficial and in such I would now endorse it. There may be and are instances in which neither the ocean nor the subsequent Nauheim treatment can be taken, by reason of considerations of time and money. In such instances I should be inclined today, in view of what I have observed and know myself and because of observations of other competent, trustworthy clinicians, to endorse the artificial Nauheim baths and resistant movements—carefully, judiciously carried out. But this, in my judgment, is a *sine qua non*, and I say this because I believe I have seen injudicious treatment, even at the spa itself, productive of anything but what I considered desirable results. If after a few days of the bath treatment the patient is not improved, it is better to return for a while to treatment by complete rest. The after-cure treatment of Nauheim, which consists in patients being sent to the Black Forest or some portion of Switzerland, is very important. Here it would be advisable to send patients for a while, if in summer, to the Adirondacks, Catskills, or White Mountains, always bearing in mind that a moderate elevation should be selected at first, to avoid possible heart strain. James M. Anders,[19] of Philadelphia, is not convinced that the home treatment of Nauheim may be carried forward with much success, for lack of the daily care and watching of a heart specialist.

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[9] Amer. Med., October 17, 1903, p. 625.

In some of these cases of dilated left ventricle following influenza, in which the heart continues rapid and irregular, and the arterial tension remains low, a prolonged course of moderate, or even small, doses of digitalis is undoubtedly useful, and I may say curative.

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[10] N. Y. Med. Jour., December 3, 1904, p. 1099.

As Lees[11] has shown, in hearts merely weak digitalis is of little value, but when we have concomitant dilatation it is often most helpful and preservative.

ACUTE CARDIAC DILATATION OF NERVOUS ORIGIN.

From experiments upon animals in which the spinal cord or the vagi have been cut it appears to be shown that in warm-blooded animals digitalis acts efficiently through the nervous system, and not entirely and directly upon the muscular fiber of the heart.

Recently, I tried[12] to show how frequently dilatation of the heart occurs as a sequence of lowered nerve tone. The examples I recorded were taken from cases which had developed slowly and after a long period of various morbid symptoms. There are other examples, however, in which the nervous breakdown comes suddenly or rapidly, and the heart is the organ apparently most affected, or at all events the one which occasions the larger number of distressing symptoms. From one day to another some of these patients become conscious they have a heart, by reason of palpitation and a feeling of weakness and inability to do their usual work, which they explain to themselves with much difficulty. To the careful physician the history of their life reveals the efficient cause of their debility and cardiac distress.

Frequently it has been the result of long-continued mental work of a severe character, to which there has rarely been even a temporary cessation. Again, it has been the immediate result of some great and sudden affliction, or of collapsed business ventures after a short and losing fight to combat relative poverty and obscurity and alas, at times, greatly diminished prestige and honorable repute among one's fellows. Occasionally, it has been a statement of an unwise or ignorant physician who, by an affirmation in regard to the patient's heart, has occasioned nervous dread and apprehension for which there was primarily no real cause. I have known an insignificant and temporary systolic or diastolic murmur, revealed to a physician by merest accident or in an examination for life assurance, when told to the patient, bring all the phenomena which we could fairly assign to slight, or more advanced, cardiac dilatation. When, in such cases, absolute confidence of the patient is secured and little by little all fears banished, the remedial effects upon the heart action, force, and size have seemed to me evident and remarkable. Of course, in such instances, and as far as possible, rest, control of habits and exercise, suitable dietary, an occasional dose of ammonia and lavender, and very moderate doses of strychnine with pepsin and bismuth, have also been employed, and it might be urged that in all these agencies should be found what was curative quite as much as in the mere allaying of nervous apprehension. However that may be, of one thing I am absolutely confident from repeated and long-continued observation, and it is that unless these patients are dealt with by someone who has an honest and earnest conviction as to what the real cause is of rapid cardiac failure and how it can be remedied, other means are of little or no avail. Indeed, some of those frequently employed are distinctly injurious and serve merely to accentuate and increase the physical disablement. To whip up this overtired muscle with repeated large doses of digitalis, or of caffeine, when nervous control is primarily lacking and when perhaps degeneration of the vagi or cardiac ganglia may be in an incipient stage, seems to me highly reprehensible and injurious to the patient. I am not sure either that a Nauheim treatment, no matter how intelligently employed, is at all adapted to it at first. We should wait at least a few weeks and see what rest and freedom from apprehension will accomplish; afterward no doubt the carbonic baths and regulated, very mild, resistant movements may be of much value to many patients. Always, however, we should bear in mind the limitation already referred to.

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[11] Acute Visceral Inflammations p. 180.

[12] Amer. Jour. Med. Sci., June, 1905.

A cause of frequent aggravation of sudden, minor cases of cardiac dilatation may be found in the foolish and pernicious use of the coal-tar products. Phenacetin, antipyrine, and above all antifebrin do untold harm. We all are familiar with many pernicious, so-called headache powders. I have known what I have regarded as evident rapid cardiac dilatation with cyanosis, dyspnœa, palpitation, weak, irregular pulse thus occasioned, and it has required all my intelligence with the immediate use of friction to the extremities, rest in bed, heat to the precordium, oxygen inhalations, ammonia, and strophanthus, to restore these patients from a condition of fatal imminence.

ACUTE CARDIAC DILATATION DUE TO IRREGULAR MODE OF LIFE.

The public latterly, because of the columns of the daily press which have shown by appeal to health board statistics the rapid rise in the number of deaths due to heart disease, has become anxious to trace out the causative influences and conditions. I have endeavored to give some and to show how we should regard such cases. To speak of paralysis of the heart, or of sudden heart failure, is to say the least unsatisfactory to the well-ordered medical mind. I believe it is also becoming very true of the layman, or laywoman, and in this fact resides their commendable search after a better and more satisfactory explanation. Unquestionably, the hurry, anxieties, responsibilities, worries and cares of modern life, always increasing and more urgent; without doubt late hours and little sleep, with the constant noise and din of the large cities, are all causes which must be controlled or eliminated, if we may hope to prevent those many nervous breakdowns, which are largely manifested by signs and symptoms of acute cardiac inadequacy depending upon dilatation. And still there are other factors to be studied and remedied. Among these we should cite the habit of too much eating and drinking; too rapid meals, meals improperly cooked and selected. Digestive difficulties, or almost sudden arrest of digestion, are brought about by stoppage of functional powers of the stomach due to strain, nervous exhaustion, supplemented or preceded by these other evident causes. How often do we see accounts of death from so-called "acute indigestion?" What does this mean? Sometimes the meal has been too abundant, too rich in varied foods with composite sauces, backed up with various wines, and no doubt what follows partakes in a measure of the exhaustion which has already touched the heart. The stomach dilates and fills with gas, due to unhealthy fermentations or the development of ptomains when the food is tainted or spoiled. Here, then, through mechanical pressure, on the one hand, and absorption of digestive poisons, on the other, are efficient causes for great and rapid weakening of a heart muscle which is already relaxed and under tone through previous causes at work. If the stomach empty itself spontaneously, or if the effort of vomiting be artificially produced, it will occasionally very greatly relieve an imminent condition. When this does not occur naturally, or the heart is so weak and fluttering that we dread to bring it about, we must depend at first upon revivifying agents like atropine, nitroglycerin, and strophanthus, which will enable the weak heart to regain at least partial control of its functions. Sometimes we utterly fail, and death, indeed, may occur before we have reached the patient and begun our active efforts in behalf of human life. In many such instances there may already be present evidences of previous arterial changes and organic heart disease; still it is very clear from the account we obtain of the history of the patient from himself or friends, that previous to the occurrence described he has always enjoyed thoroughly good health, as far as he knew. At all events, he had never suffered at all from symptoms of heart disease. It is sudden or rapid dilatation of the heart which causes the death and not the chronic condition revealed at the autopsy. This he might very well have carried for months and years without suffering, and, indeed, without knowledge of it, unless informed of its presence accidentally as it were. The great and almost insuperable difficulty is to declare certainly and positively at the autopsy what physically in the heart has been brought about within a few brief hours or days previous to death, and what belongs altogether to lesions of an older date.

Just as I have directed attention to the fatal effects at times of injudicious overeating and drinking, in like manner I would emphasize the extreme folly of persons near or past middle life running to catch trains, going up stairs rapidly, playing active tennis, lifting heavy persons or weights beyond their powers, and thus occasioning heart strain. The acute dilatation of the heart may be accomplished in a few minutes. It may occasion immediate death, or future invalidism which is never entirely recovered from. The old saying that a man is as old as his arteries is doubtless true, but physiologically we cannot gauge accurately in every case arterial changes. We do know, however, that doing certain things involves great risks to the city man of many cares and responsibilities, who has had no previous daily healthful training which would enable him to resist successfully sudden or prolonged physical effort. Sudden strain of short duration is frequently very injurious and at times fatal. When excessive and more prolonged, the attendant risks of more marked and irremediable acute heart dilatation are in the aggregate more considerable. What is true and of great moment to almost every man and woman of middle life—and absolutely prohibitive with greatly advanced years—should also be borne in mind and guide us as regards rational advice to give young men and girls. I have seen at least one young man of slight build, who was in the contest of a running match and who suddenly collapsed from so-called heart failure. He had been in fairly good previous training, but the pace and distance were finally too much for him, and he dropped in his tracks. I cared for him until his heart got back some strength and resumed its functions sufficiently for him to sit up, and he was then carried to his home. Such a case is one of acute heart dilatation. I have been called to the ball-room where a young woman, after dancing and late hours for several successive nights, dropped exhausted and panting on the floor and was carried to her bed-room. Here, through stimulants, mustard locally, ammonia to the nostrils, dorsal decubitus, loosening and removal of corsets and all constriction from petticoats, garters, slippers, etc., there was slow recovery, and the attack of syncope was dissipated. Such faint turns must, in my judgment, usually be translated simply by calling them attacks of acute cardiac dilatation. All hospital physicians are familiar with other instances —those in which after a drunken bout, or a day of excessive strain, or heavy lifting on the part of a truckman or longshoreman, in which, also, there is a history of repeated daily drinks of whiskey, the patient has laboring, rapid, stertorous breathing, cold cyanosed nose, lips, and fingers, clammy frontal perspiration, frequent, almost imperceptible pulse, and moist mucous rales throughout the chest, showing acute pulmonary œdema.

At the same time we find notable cardiac dilatation, especially of the right heart, with distended throbbing jugulars, pronounced epigastric pulsation, and diffuse, feeble, irregular, and intermittent cardiac impulse. In these cases immediate venesection followed by nitroglycerin by the mouth, $\frac{1}{100}$ grain every half-hour for a while, hypodermic injections of atropine, $\frac{1}{60}$ grain three or four times in twenty-four hours, and repeated dry cups all over the chest, followed by oxygen inhalations, will alone save life many times.

"Among these cases, however, are unquestionably some in which the physical signs of cardiac dilatation are impossible to determine accurately. I can well understand that their existence should be denied. In place of such a diagnosis I cannot but substitute one of loss of nerve power, either in the intracardiac ganglia or in the trunk of the vagi. To admit this would be, perhaps, also to acknowledge that the heart muscle was intact and the cavities of normal dimensions. Such a belief would be strengthened by those instances in which certain cardiac tonics, especially digitalis, are of little apparent value, perhaps, indeed, directly injurious, and rest in bed and suitable liquid diet with alcoholic stimulants appear to be most useful."[13]

The acute dilatation due merely to the use of tobacco, when excessive and long continued, is, I believe, often recognizable, although not as readily detected as in the other instances already described. The tobacco heart is often a cause of very great distress to the patient on account of heart flutterings, throbbings, irregular pulsations, coming on at times when the patient is in absolute repose, and making sleep wellnigh impossible. Such patients become specially self-centred, timorous, fearful, and anxious. Whatever else is done, tobacco must be given up, and if indulged in at all, must be taken in the mildest form and most moderate quantity. Usually, however, it is best to give it up entirely for several months, or until complete re-establishment of health has taken place. This will almost certainly occur ultimately, provided no organic heart disease already exists in advanced form.

In many cases of acute dilatation of the heart, it may be difficult to pronounce absolutely that it exists, because of the presence of pleural effusion, pulmonary solidification, or emphysema. In these cases we are often led into error, either to believe that more dilatation is present than really exists, or, on the other hand, to deny its existence when after-events prove it to be there. There is no question that light, accurate, and very careful percussion with a single finger, so well emphasized by Lees, is the surest way to avoid serious mistakes. Deep percussion with too vigorous action and several fingers, or by means of hammer and pleximeter, will often bring out resonance from other organs and conceal the correct and accurate limitations of the cardiac margins, and hence the size of the ventricles and auricles, and indeed of the entire heart.

It was stated authoritatively that during one week of June, 1906, 125 deaths occurred from heart disease in New York City. Further it was affirmed that the percentage of deaths from this cause had notably increased during the last few years. The proportion, indeed, had risen from 18 in 10,000 in 1880 to 30 in 10,000 in 1904. This steady increase of mortality may well cause reflection. In some cases death came very suddenly, in others after a period of confirmed invalidism. No doubt in many instances of both kinds the fatal occurrence would have been indefinitely postponed, if rest and relaxation from the cares of business had been taken sooner. Unfortunately, the realization of this often comes too late, when the calamity has occurred. Much of this lamentable outcome could be avoided if men in important positions would consider their vital energies as carefully as their various daily occupations. If short vacations were taken from time to time, there would be far less need of long absences from affairs to recuperate. It is only too true that what in disease has taken a long while to develop, requires an equally long time to become corrected, so as to get back one's original health and strength.

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[13] Essays on Clinical Medicine. Beverley Robinson, M.D., 1903, p. 86.

One great difficulty in estimating the risk of an ultimate breakdown proceeds from the fact of the insidious march of the physical changes which produce the disastrous results. Too often indelible marks of continued work and effort are left before a prolonged and proper vacation is taken, and then it is too late to reap much benefit from it. This holds good of the vacation of travel, or a change to the woods, an ocean voyage, or a cure at some well-known spa. The moral of all this is to take work reasonably and quietly, and it means happiness and prosperity; take it in a contrary way, and it means disease and death. The plea is for more time to eat and sleep; less hurry and worry; readiness to take needed rest when weary; more satisfaction in slower and surer gains; and the abandonment of the attempt to attain the impossible. Let us take time to apply to ourselves the lesson of 125 deaths in one week in New York City.[14]

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[14] Editorial in New York Herald, June 14, 1905.

EXPERIMENTAL STUDIES OF CARDIAC MURMURS.

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In connection with experiments on the production of valvular lesions which have been carried on by one of us[1] during the past year, we have made a series of observations on the cardiac sounds with especial reference to transmission of murmurs in the dog's heart. Having studied the character of the heart sounds of the normal dog we have produced, as far as possible, the lesions commonly observed in man, each in several cases. In other experiments we have made a few observations concerning the development of functional basic murmurs. In the present communication it is our desire to refer to some of the more interesting points which have been brought out by that work which we have already done.

Methods. After etherization, the anterior part of the chest is removed, artificial respiration established, the pericardium opened, and the heart thoroughly exposed.

A special stethoscope (Fig. 1) has been devised, the bell of which is of small diameter (1 cm.), so that the sounds heard over a small organ may be better localized, and long (6.8 cm.), so that it may be placed readily upon the rather deep lying structures.

Position of the Heart. The arrangement of the chambers of the dog's heart when exposed *in situ* is somewhat different from that of the human heart. A much larger part of the anterior surface of the organ is occupied by the left ventricle, while the right auricle occupies a smaller area in the upper right-hand corner, and the tip of the appendage of the left auricle is usually visible below and adjacent to the pulmonary artery, just as it is *not*,[2] as a rule, visible in the human heart despite the orthodox illustrations.

Normal Heart Sounds. The heart sounds of the dog are closely analogous to those of the human being. The first sound over the left ventricle is duller and more muscular; over the right ventricle somewhat more flapping and valvular. With the heart exposed, it is possible to put the stethoscope directly over the aortic and pulmonic rings. The difference in the character of the second sounds, which is so often to be detected in the human being, is here very striking. The second sound at the aortic orifice is of very much greater intensity than that at the pulmonic ring, of a more ringing and musical quality, while the second sound over the pulmonic valves has a deader, more wooden character. This difference is marked and characteristic, so much so as to confirm me (Thayer) in the conviction which I have often expressed, that it is sometimes possible, in the human being, to determine the origin of the second sound to which one is listening, by its character alone—a point which may be of importance in cases in which the second aortic sound is heard best to the left of the sternum.

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[1] MacCallum, Johns Hopkins Hosp. Bull., 1906, xvii, 260.

[2] It should be noted, however, that in some dogs an auricle which was evident *intra vitam*, disappears *postmortem*. Similar conditions may exist in man.

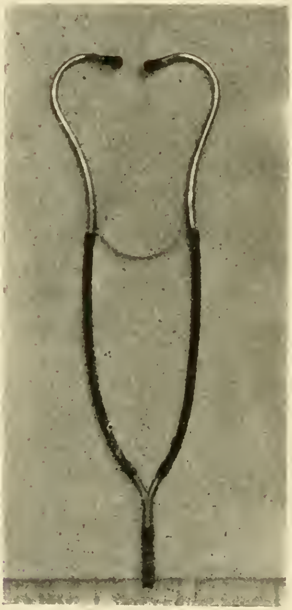

Fig. 1.—Stethoscope with a long bell of small diameter for auscultation of the dog's heart.

Aortic and Pulmonic Stenosis. In our first experiment we produced aortic and pulmonic stenosis. This is a perfectly simple procedure, carried out by the passage of a cord about the vessel just above the valves. This may be tightened to produce any degree of stenosis desired. The production of a stenosis causes the characteristic thrill and rough murmur transmitted upward in the course of the current, just as is observed in man. The experiment differs in no essential point from that which may be performed with a rubber tube attached to a faucet.

A striking and particularly interesting point is the ease with which murmurs may be produced in the pulmonary artery. Very slight pressure over the conus arteriosus or over the artery results in the development of a systolic thrill and murmur immediately beyond the point of pressure. The ease with which these murmurs are produced by pressure suggests immediately that a similar process may account for some of the frequent basic pulmonary murmurs which are heard in man. If systolic murmurs can be produced in man by a degree of pressure over the conus arteriosus as slight, relatively, as that necessary to bring about the same phenomenon in the dog, it is but natural to assume that the resistance offered by the chest wall alone may, in many instances, result in systolic murmurs in the pulmonary area.[3]

Aortic Insufficiency. In eight instances (experiments I, II, IV, V, VI, VII, X, XII) an aortic insufficiency was produced. In all cases a loud diastolic murmur developed at the aortic ring; this murmur sometimes replaced the second sound; sometimes the two sounds were heard in association.

Always loudest at the aortic ring, it was transmitted a short distance upward along the aorta, but best downward toward the ventricle. Over the ventricle the murmur was always much feebler, and in some instances it was inaudible at the apex. In some cases it was heard loudest on the anterior surface of the ventricle at about the middle of the line separating the right and left ventricles; in other instances, it was more distinct around toward the left, just below the pulmonic valves. Sometimes it was feebly audible through the right ventricle. The character of the murmur was exactly the same as that of the souffle of aortic insufficiency in the human being. Especially characteristic was the softness of the sound over the ventricle. The first sound over the left ventricle became dull and humming, just as in aortic insufficiency in man.

In several cases in which, after the production of extensive lesions, the ventricle became dilated and feeble, the murmur diminished greatly in intensity, sometimes disappearing wholly over the ventricle, although still faintly audible at the aortic ring.

In three of these eight cases a soft systolic murmur appeared also at the aortic valves. Marked pulsation of the peripheral vessels occurred just as in man, resulting, in some cases, in an exquisite hammer pulse, with a pistol-shot tone in the femorals.

In one case in which we were studying the basic murmurs in the heart of an animal which had been bled repeatedly and afterward infused with salt solution, there developed an exquisite aortic insufficiency with a double (systolic and diastolic) murmur at the ring and over the left ventricle which was visibly dilated. Later a typical murmur of mitral insufficiency, associated with pulsation of the left auricle was to be made out. A sharp second sound was also audible at the aortic ring. The accompanying tracing (Fig. 2) shows, in an interesting way, the difference between the character of the femoral pulse curve before and after the development of aortic insufficiency. Unfortunately, the cannula in the left auricle had become clogged so that the pulsation of the blood in this cavity with the onset of mitral insufficiency was not recorded. The necropsy, in this case, showed that the valves were intact, although subendocardial hemorrhages in both ventricles were probably evidences of the distention to which the walls had been subjected by the artificial plethora.

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[3] In the discussion upon this communication, Dr. Janeway referred to the frequency of pulmonic systolic murmurs during forced expiration. May it not be that this phenomenon is due in part, at least, to the fact that, by the retraction of the lung, that which normally acts as a cushion between conus and chest wall is removed?

The most interesting points brought out by these cases of aortic insufficiency would seem to be the intensity of the diastolic murmur at the ring and its feebleness over the aorta above and over the right and left ventricles below, a condition in every way analogous to that observed in the human heart, and wholly in accordance with the well-known fact that the murmurs of aortic insufficiency are often very difficult to hear. Especially interesting was the production of a functional aortic insufficiency from dilatation, an observation supporting entirely the conception of those observers who have insisted upon the important part which the muscle of the left ventricle plays in the closure of the aortic ring.

Mitral Stenosis. In two cases (experiments VII and IX) attempts were made to produce a mitral stenosis. It is needless to say that it was impossible to bring about changes in the valve analogous to those in a valvular stenosis in the human being. The nearest approach that was made to this was to apply a clamp at about the position of the mitral ring. The tightening of such a clamp resulted in great distention of the left auricle, rise in pulmonic pressure, and an immediate increase in the intensity of the second pulmonic sound, with a corresponding enfeeblement of the second aortic sound. In one case it was possible, at a certain point of rather moderate tightening of the clamp, to detect a slight presystolic murmur, heard best midway between the clamp and the apex, over the left ventricle. This murmur disappeared with further tightening or with relaxation of the clamp. In the other case no murmur could be produced. The first sound instead of becoming snapping and valvular, as in the characteristic valvular stenosis, became duller and more humming.

Mitral Insufficiency. In six cases mitral insufficiency was produced by tearing the valves or chordæ tendineæ with the knife-hook described elsewhere,[4] which was passed through the auricle or through the carotid (experiments II, IV, VI, X, XI, XII). The production of a mitral insufficiency resulted in the development of a blowing, rather soft, systolic murmur heard with moderate intensity

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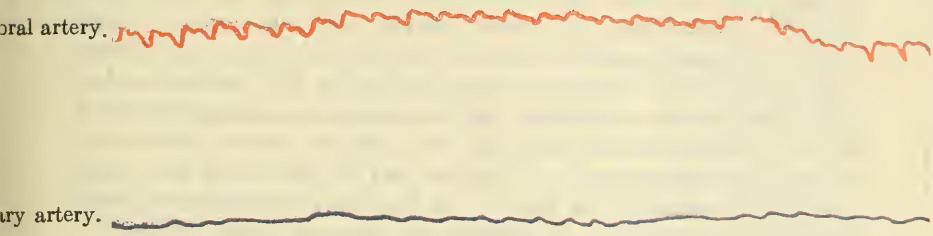
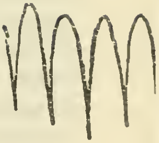
[4] MacCallum, loc. cit.

Infusion continued.





over the left ventricle. This murmur replaced the first sound wholly or in part; its general character was closely similar to the analogous murmur in man.

In the dog's heart, however, it is possible to place the stethoscope over the left auricle just beyond the valves at the point at which, theoretically, the murmur should be most intense. This is, indeed, the case, the murmur in most instances being very loud and associated with a thrill of great intensity. The murmur was, in some instances, transmitted to the pulmonary veins of both sides; in one case it was audible over the back of the animal. In one case, with distention of the ventricle, a characteristic murmur of mitral insufficiency developed as a result of dilatation of the ring.

It would greatly simplify the teaching of auscultation of the heart if it were possible to make demonstrations such as the foregoing before the student. For years one of us[5] has been in the habit of employing a very simple mechanism, taught him nearly twenty years ago by a valued teacher[6]—a rubber tube attached to a faucet of running water. By producing any degree of pressure upon the tube at a given spot, and by increasing or diminishing the rapidity of the flow of the fluid, many points of importance with regard to the origin and transmission of cardiac murmurs may be illustrated. Indeed, a student who is really familiar with the anatomy of the heart and surrounding organs, and with the character and theories with regard to the origin of the heart sounds, can, after such a demonstration, work out the diagnosis of the simpler valvular lesions with little assistance. The very simplicity of the demonstration sometimes leads the student to doubt its complete applicability to conditions existing in the living animal. For years I[7] have been in the habit of teaching students that the murmur of mitral insufficiency is doubtless loudest at the mitral ring and just beyond this point in the left auricle—that its intensity at this point is probably great—that there is probably also a well-marked thrill over the left auricle—that the deep position, however, of the left auricle and the mitral ring render these conditions undemonstrable—that the murmur is audible at the apex alone because this is the only point at which it is possible to approach the left ventricle—that the murmur of mitral insufficiency is not, as a rule, very intense because, as in the experiment with the rubber tube, the murmur and thrill diminish rapidly as one passes above the point of constriction. But obvious as such an explanation may seem, it is often difficult to impress the student. What a luminous demonstration it would be if one could supplement the experience with running water with experimentally produced lesions as above described—if it were possible for the student to put his finger and his stethoscope on the left auricle as well as on the apex of the heart.

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[5] Thayer.

[6] Dr. W. W. Gannett, of Boston.

[7] Thayer.

There is no reason why such demonstrations could not be made to small sections of students. We are daily inculcating in our students a wholesome skepticism with regard to all things that are insusceptible of proof; and wherever palpable confirmation of our theoretical explanations of morbid phenomena can be offered, we should not fail to produce it.

In one case of dilated heart a characteristic murmur of mitral insufficiency developed as the result of a dilatation of the ring.

Protodiastolic Gallop Rhythm and Murmur. In one instance (experiment IX) an interesting phenomenon was observed. This experiment was started with the idea of observing whether, with aortic insufficiency, the pressure in the left auricle was increased without coincident mitral insufficiency. Cannulæ were inserted into the left femoral artery, into the appendage of the left auricle, and into the peripheral end of a pulmonary artery. By so doing the heart was rotated over toward the right, while the left auricle was held rather tensely in position by the cannula in the auricular appendage. This appeared to have the effect of placing the heart in a position in which it was, in a way, suspended by the left auricle. There then became audible over the left auricle and just below the valves a first, a second, and then, after a momentary pause, a third sound—a diastolic rumble in every way similar to the early diastolic murmur heard in some cases of mitral stenosis. When the heart became more rapid this rumble extended into presystole, simulating, closely, a mitral stenotic murmur. There was no mitral regurgitation. Gradually the murmur disappeared and one heard only a thud, which became clearer with the decreasing rate of the heart's action. One was able to observe the systole of the ventricle immediately followed by the second sound, which occurred while the ventricle was still in a state of contraction. Then followed the sudden dilatation of the ventricle which occurred *abruptly, with a distinct shock, having altogether the appearance of an active muscular process.* Synchronously with this shock occurred the third sound. This thud was clearly analogous in time (*a*) to the so-called *opening-snap* heard in mitral stenosis and ascribed to the sudden tension of the thickened mitral valves with the beginning of diastole. Its character, however, was precisely that of (*b*) the sound in protodiastolic gallop rhythm heard under so many circumstances, especially in nephritis, dilated heart, adherent pericardium, etc., and ascribed variously to the impact of the auricular blood upon a ventricle which has lost its elasticity (Potain),[8] or to an active muscular dilatation of the ventricle (Brauer).[9]

The case is interesting because we were able to *see*, so to speak, the association of this sound with a definite moment in the heart's cycle—that is, the shock of what appeared to be an active diastole, and the association of a murmur with conditions which seemed to suggest the suction of blood into the ventricle.

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[8] Clinique médicale de la Charité, 8°, Paris, 1894, 27.

[9] Verhandl. d. XXI Cong. f. inn. Med., Wiesbaden, 1904, 187.

It may be well here to mention the fact that the presence of a third sound at just this period of the heart's cycle is not infrequent in the apparently normal heart, especially in children. The sound is sometimes a dull thud, sometimes merely a hum, and occurs shortly after the second sound, that is, at the beginning of ventricular diastole. Sometimes it is followed by the faintest suggestion of an echo, so that I[10] have long been in the habit of maintaining that the entrance of blood from the auricle into the ventricle through a normal orifice is associated in some instances with a slightly audible sound.

Basic Systolic Murmurs. In four cases (experiments III, VIII, IX, XI) we have studied the effect of changes in the heart's action on the sounds at the arterial orifices. The question here is too complicated to justify elaborate discussion on the basis of our few experiments, but a brief summary of what we have observed may be of interest.

A. *Aortic Murmurs.*

1. Systolic murmurs at the aortic ring were never found in the normal heart.
2. Variations in arterial pressure produced by nitroglycerin or adrenalin did not result in their appearance.
3. These murmurs appeared in conditions of rather low pressure.
4. In two instances (III and XI) they followed bleeding alone. In such cases the administration of adrenalin with a rise in pressure caused the disappearance of the murmur.
5. Systolic aortic murmurs were especially frequent after infusion of salt solution following bleeding.
6. In general, aortic systolic murmurs were especially frequent after bleeding followed by infusion, in association with what appeared to be rather abrupt action of the ventricle and rather large excursions of the pulse. They sometimes followed hemorrhage alone. They never appeared as the result of modifications in pressure following the administration of nitroglycerin or adrenalin.

B. *Pulmonic Murmurs.*

1. As has been already noted, pulmonary systolic murmurs may be produced with great ease by slight pressure on the conus arteriosus or the pulmonary artery.
2. In two instances a pulmonary systolic murmur developed after considerable hemorrhage.
3. In one instance with a low general pressure following nitroglycerin, a pulmonary systolic murmur developed. The adminis-

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[10] Thayer.

tration of adrenalin and nitroglycerin had, however, but little effect in modifying the pressure in the pulmonary artery.

4. The administration of adrenalin resulted in an increase in systolic pulmonary murmurs when present, during the first strong beats which followed, but during the period of very high pressure occurring in the general circulation, the pulmonary murmurs as well as murmurs at the aortic area, tended to diminish, returning, however, again, if present before, with a fall of pressure at a period when aortic murmurs still remained absent.

5. Infusion of salt solution resulted commonly in the development of systolic pulmonary murmurs.

6. In a general way, conditions which resulted in especially abrupt action of the right ventricle with a large excursion of the pulse wave in the pulmonary artery seemed to favor the development of systolic murmurs at the pulmonary orifice, especially if there had been a preceding hemorrhage followed by infusion of salt solution.

Murmurs were produced more readily in the pulmonary artery than in the aorta, notwithstanding the very slight variations in pressure which occur in the lesser circulation. This was probably due, at least in part, to the fact that with a more or less indistensible ring, the pulmonary artery beyond is much less resistant than the aorta, and capable of greater dilatation with ventricular contractions of moderate force, thus producing, just beyond the pulmonary ring, those conditions favorable to the development of fluid veins, and consequently a thrill and murmur.

In general, our observations seem to show that a change in the character of the circulating blood, such as that which results in the replacing of a considerable quantity of blood by salt solution (diminished viscosity?)is extremely favorable to the development of systolic murmurs at the base.

In conclusion, it may not be amiss to repeat that we have been strongly impressed, during these studies, with the feeling that where auscultation is taught in small sections, several exercises in the study of experimental cardiac murmurs in the dog would do more toward making clear in the mind of the student the essential facts, without which rational diagnosis of cardiac lesions is impossible, than any other method of instruction with which we are familiar.

ANEURYSM OF THE ARCH OF THE AORTA: RUPTURE INTO THE SUPERIOR VENA CAVA.[1]

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The patient the subject of this report was admitted to St. Mary's Hospital, Philadelphia, October 19, 1905. He was an Irishman, aged fifty-four years, by occupation a weaver.

Previous History. He had typhoid fever forty years ago and rheumatism twenty-four years ago. He denies syphilis. His wife died a number of years ago, never having had any miscarriages. He drinks considerable whiskey, and was drunk two weeks before his admission to the hospital. His parents are dead, of unknown cause. He has five brothers and sisters living and well.

Present Illness. He was perfectly well until October 17, 1905 (two days before admission). At that time he noticed a sudden swelling of the face and arms, accompanied by cough and dyspnœa, which prevented him from lying down in bed. He had no pain. Before this attack he worked every day and never noticed any dyspnœa, œdema, or palpitation.

Examination on Admission. The patient suffers from orthopnœa. His face, both arms, neck, and upper chest, are swollen to twice their normal size, and are œdematous and markedly cyanosed. All of the superficial veins are greatly enlarged; some are as large as a crow-quill. The external jugular vein on the left side is dilated to the size of the index finger, and a branch of this vein crosses the superior part of the sternum and anastomoses with the mammary veins. The veins at the margin of the ribs form great varices; in places the chest is almost black from the number and size of these veins. The superficial veins over the left abdominal wall are slightly increased in size. The hands, the legs, and the genitals are not swollen. There is slight œdema of the abdominal wall. The heart dulness, as shown in Fig. 1 *A*, is about normal in size. The apex-beat is difficult to palpate and is quite irregular. The sounds at the aortic cartilage are clear and the second is accentuated. There is an indistinct "crunch" at the xiphoid. At the apex the first sound is weak and valvular and there is a very indistinct systolic murmur. No abnormal dulness is made out over the sternum. There were no murmurs in the vessels or at the root of the neck. The systolic blood pressure in the left arm is 189, in the right arm 160; diastolic pressure in the left arm 160, in the right arm 120. There is a slightly dull note at the apex of the right lung. There are no other abnormal percussion sounds. The breath sounds are normal throughout. There is no abnormality in the abdomen; neither the liver nor spleen can be palpated.

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[1] Read at the meeting of the Association of American Physicians, Washington, D. C., May 15 and 16, 1906.

October 22. Patient complains of great dyspnœa and troublesome cough. The cyanosis and œdema of the upper half of the body have increased. There is marked dulness on percussion under the first piece of the sternum, as shown in Fig. 1 *A*. No pulsation can be felt over this area or in the suprasternal notch. There is a slight systolic murmur over the dull area. Auscultation of the trachea does not develop compression of that organ. At the apex of the left lung the expiration is slightly prolonged. Tracheal tug cannot be developed. There is no pulsation over the area of dulness. A radiogram was taken and did *not* show the presence of an aneurysm.[2]

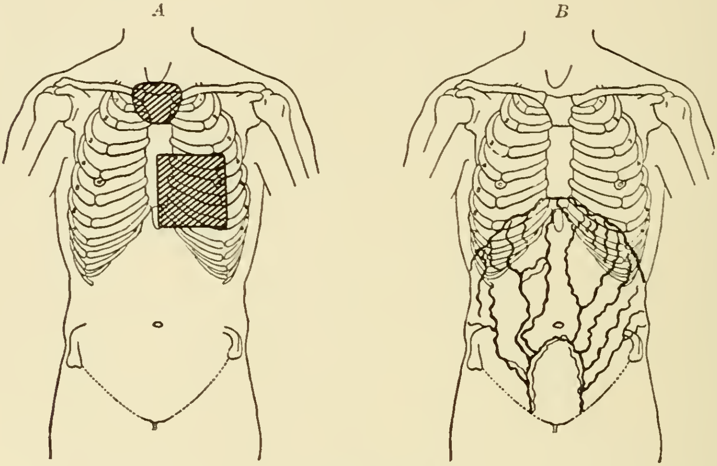

Fig. 1.—*A*. The area of heart dulness under the sternum. *B*. The distention of the abdominal veins.

23*d*. Patient has great distress upon swallowing, on account of the dyspnœa it causes.

25*th*. Laryngological examination shows venous engorgement of the mucous membrane, but no signs of pressure.

26*th*. Dulness over the first piece of the sternum still persists. There is no visible or palpable pulsation. At the junction of the first and second pieces of the sternum there is a marked to-and-fro murmur with a very sharp second sound. Pressure over the sternum causes great distress in breathing.

31*st*. The apex of the heart is far to the left of the nipple line. There are signs of pleural effusion in both chests. About one pint of bloody liquid was removed from the right chest. A marked dermatitis has developed in both arms.

November 12. The veins over the chest are more distended. No dulness can be made out over the manubrium, but the marked double murmur with a sharp second sound can be heard. It can also be heard in the back. There is marked distention of the abdominal veins.

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[2] This, of course, was because of the small size of the aneurysm, immediately under the sternum.

26*th*. The abdominal veins are greatly enlarged (Fig. 1 *B*). These veins anastomose with the greatly distended veins of the chest. There is no marked enlargement of the veins on the posterior part of the chest. The dulness over the sternum is marked. No thrill or pulsation can be felt. The same murmur, double in character, can be heard, with an accentuated second sound. There is no murmur in the veins of the neck.

The patient died suddenly on the morning of October 27. There were slight rises of temperature during the course of the disease. The urine was normal. The hemoglobin was diminished, but there were no other blood changes.

Autopsy. The body is that of a middle-aged man. There is no œdema of the extremities. The superficial veins of the chest and abdomen are tortuous and considerably dilated. On removing the sternum the azygos veins are seen to be greatly distended. The right pleural cavity contains about 950 c.c. of cloudy amber fluid. The parietal veins are dilated. The left pleural cavity contains about 135 c.c. of cloudy amber fluid. The parietal veins are distended. At the apex of the right lung are a few healed scars; scattered throughout the upper lobe are numerous white areas, about the size of a millet-seed, which on section are seen to be miliary tubercles. In two or three places these tubercles are massed together and are beginning to caseate. The left lung is smaller than its fellow. The surface is of a very dark purple color. The lung is nowhere crepitant except at the anterior margin. The cut surface presents a firm, marbled appearance; the alveoli appear to be obliterated; on compressing the lung a white, purulent fluid exudes. At the apex there are several old, healed scars; there is no evidence of acute tuberculous involvement. A section of the lung sinks in water. The pericardial cavity contains about 50 c.c. of turbid amber fluid. The surfaces are smooth.

Heart and Aortic Arch. The heart lies in its usual position. It extends on the right about 5 cm. to the right of the median line. The displacement to the right is due to the immense effusion in the left pleural cavity. Just beneath the sternum, on a level with the third rib, extending from the second rib to the third interspace, there is an indefinite mass bound about by adhesions, but not attached to the sternum. The heart itself is approximately normal in size. The muscle is dark-red in color and somewhat decreased in consistency. The walls are thinner than normal: the right ventricle averages 1 mm. in thickness, the left ventricle about 10 mm.; the cavity is about normal in size. The leaflets of the tricuspid, pulmonary, and mitral valves are thin, delicate, and apparently normal. Two leaflets of the aortic valve contain a calcareous nodule at their bases which protrude into the sinus of Valsalva. They measure about 3 mm. in diameter; otherwise, these leaflets are thin and delicate. All the valves are apparently functionally intact. The circumference of the aortic ring is 7 cm. Scattered over the various portions of the arch, as well as of the thoracic aorta, are numerous atheromatous plaques, measuring from 2 to 17 cm. in their greatest diameter; none of them are actually calcareous. At a distance of 5.5 cm. from the aortic ring is a sacculated dilatation of the ascending part of the arch of the aorta, measuring 3.5 cm. in diameter (Fig. 2). This sac, which protrudes from the aorta on the right and slightly posterior, measures 3 cm. in depth, and is narrower than the greatest diameter of the aorta itself. The vena cava is constricted near the centre, where there is a small aperture about 7 mm. in diameter, and with a smooth edge, so that there is direct communication between the ascending aorta and the superior vena cava (Fig. 3). The aneurysmal sac distends the aorta at the origin of the innominate,

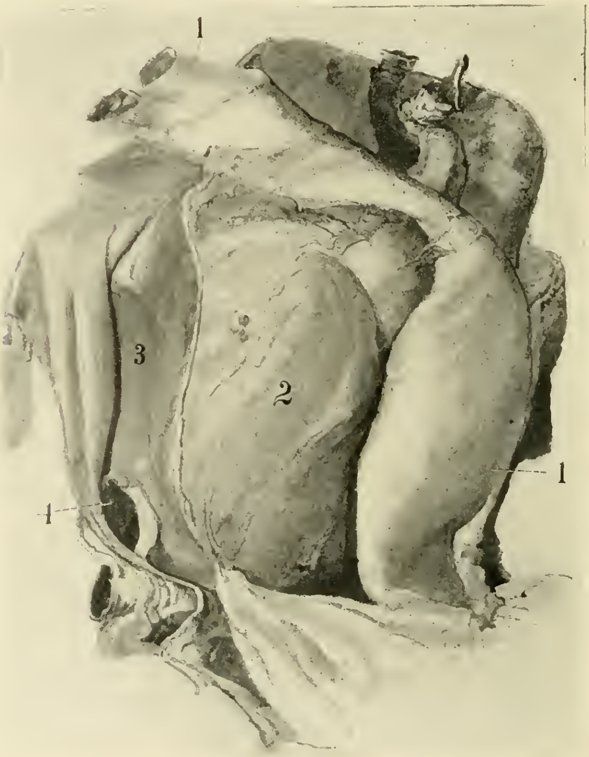

Fig. 2.—Exterior of the aneurysm, showing rupture into the vena cava; the vena cava laid open. 1. Aorta, exterior surface. 2. Adventitious sac filled with clot. 3. Vena cava laid open. 4. Opening from the sac into the vena cava.

but the aorta is apparently normal at the origin of the left carotid artery. The aneurysmal sac has ruptured downward and forward, and has formed an adventitious sac between the aorta and the superior cava. This adventitious sac has for its walls the ruptured aorta, the connective tissue surrounding the vessel, the superior cava, and the pulmonary artery. It measures from above downward 6.5 cm., and is approximately 3.5 cm. in diameter. It has compressed the superior cava 3 cm. above its entrance into the auricle, and has ruptured into the cava at this point. The opening into the cava is about 7 mm. in diameter and is elliptical in outline, the long axis of the ellipse being in the direction of the vessel. The true aneurysmal sac in the aorta is entirely free from clot, but the adventitious sac is filled with a firmly organized coagulum. The blood has forced its way between the adventitious sac wall and the organized clot. It is seen from this that the actual rupture of the true aneurysmal sac must have occurred at a time antedating the symptoms, but the rupture from the adventitious sac into the cava doubtless occurred at the onset of the symptoms.

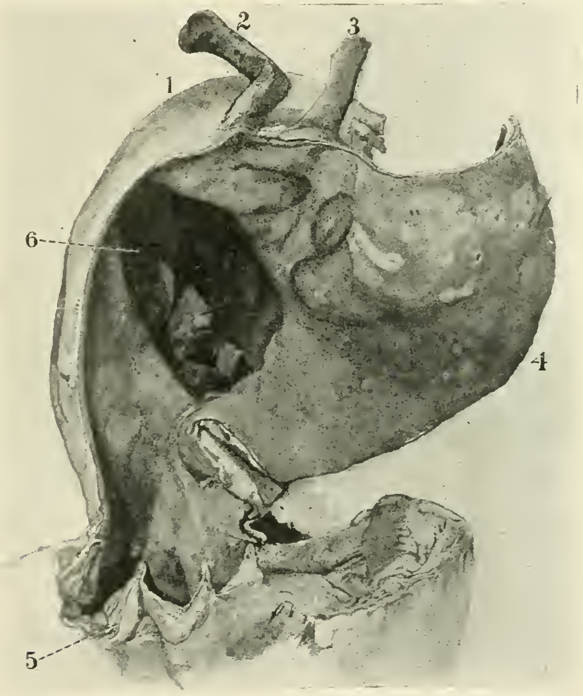

Fig. 3.—Interior of the aorta, showing the aneurysm. 1. Innominate artery. 2. Left carotid artery. 3. Left subclavian artery. 4. Aorta. 5. Aortic valves. 6. Aneurysm with clot at the bottom.

The liver weighs 1150 grams and measures 20 x 17 x 6 cm. The surface is smooth, free from adhesions, and of a dark purplish color. Near the falciform ligament on the surface of the left lobe is a hard, calcareous nodule, about 5 mm. in diameter. The cut surface is brownish red in color. The central veins are clearly seen and are surrounded by a paler area of a light-brown color. Glisson's capsule is not increased. The consistency of the organ is diminished and the organ may be readily torn. The gall-bladder is normal. The spleen weighs 50 grams and measures 6 x 7 cm. The bloodvessels project above the surrounding pulp. There is a slight increase in the trabeculæ.

The right kidney weighs 105 grams and measures 10 x 6 x 2.5 cm. The left kidney weighs 95 grams and measures 6 x 2.5 cm. Their capsules strip with difficulty. The external surfaces are of a dark purplish color. On section the cortex averages 7 mm. in thickness. The consistency of the organs is increased. The Malpighian bodies are not visible.

The pancreas, stomach, and intestines are apparently normal. There is moderate atheroma, with a few calcareous plates in the abdominal aorta. The walls of the urinary bladder are somewhat thickened and congested.

Histological Examination. Heart. The epicardium is wanting. The endocardium is apparently normal. Interstitial connective tissue is not increased. Fragmentation of the muscle fibers is very marked in some places, less so elsewhere. Polar pigmentation is very marked, every fiber giving evidence of it. The capillaries are slightly thickened. The striæ of the muscle fibers are preserved.

Liver. The external capsule and the capsule of Glisson are apparently of normal thickness. All the capillaries are more or less engorged with blood cells, but especially the intralobular veins. The bile ducts are normal. In many places the columns of liver cells are farther apart than usual, and in spaces thus left red blood corpuscles are to be seen. Many of the hepatic cells are filled with a brown pigment. Those surrounding the terminals of the hepatic vein are especially rich in pigment granules, while the cells in the periphery are often quite free of them. The nuclei of the cells take a deep-blue color with hematoxylin.

The rarity of this rupture of an aortic aneurysm into the superior cava may be judged by some figures by Kelynack.[3] He reports 4593 cases of aneurysm of the arch of the aorta with rupture in thirty-two cases, or 6.9 per cent. Of these, one was into the superior cava. Therefore, one would have to see in the neighborhood of 5000 aneurysms before observing a rupture into the superior cava Two cases similar to the one here reported have been presented to this Association (Pepper and Griffith[4] in 1890, and Stengel[5] in

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[3] Lancet, 1897, ii, 193.

[4] Trans. Assoc. Amer. Phys., 1890, v, 45.

[5] Ibid., 1900, xv, 532.

1900). Thus three out of five of reported American cases have been reported before this Association.

Pepper and Griffith collected all the cases of rupture into the vena cava which had been reported previously, in all, twenty-nine cases. I am now able to add seven more cases, those reported by Ord,[6] Mercudino,[7] Bruce,[8] Stengel, Rendee,[9] Leyden,[10] and my own, making a total of thirty-six cases. Careful review of these thirty-six cases leads to a confirmation of the thorough and exhaustive analysis which Pepper and Griffith made of their cases sixteen years ago. The chief points were: The sudden cyanosis, œdema, and distention of the veins of the face, neck, arms, and upper half of the trunk. The picture is so striking that once seen it can never be forgotten. The picture is almost grotesque, so great is the disparity in size and appearance between the upper and the lower halves of the body. It was my fortune to see during life the case reported by Pepper and Griffith, and while the condition had not again been brought to my mind, I was able at once to recognize my case as one of this character when first seen.

The murmur so accurately described by Pepper and Griffith, the continued high-pitched murmur with increase during systole and general decrease in intensity during the diastole, has been present in sixteen of the thirty-six cases reported. A to-and-fro murmur is described eight times, systolic six times, diastolic once, and is not mentioned five times. The symptoms and sings of an aneurysm in the chest, of course, make the diagnosis much more easy and certain.

Thirteen of the thirty-six patients gave the history of being entirely well before the symptoms of rupture; but at autopsy twenty-nine of the cases were shown to have tumors as large or larger than an orange, and therefore were probably susceptible of a diagnosis of aneurysm at or before the rupture, had the point been given attention, —although, as Osler has pointed out, there are cases of aneurysm with symptoms but without physical signs, and aneurysms with signs but without symptoms. All of the symptoms and physical signs of rupture into the cava may, however, be simulated by pressure upon the cava, as proved by the following case reported by Salomon.[11]

A man was admitted to the Hospital Laënnec, April 24, 1903. On a certain night this individual was heard making noises in his room. No attention was paid at the time. In a few hours he was found unconscious and taken to the hospital. He was comatose. There was an intense cyanosis of the face, neck, and arms. There was little œdema. There was a continuous murmur over the base of the heart, with intensification during systole and diastole. There were signs of aneurysm of the arch of the aorta. On account of the peculiar cyanosis and rapid onset a diagnosis of aneurysm rupturing into the cava was made. At the autopsy an aneurysm of the ascending portion of the arch was found. The right side of the sac was in connection with the vena cava superior. Careful examination of the vena cava and of the aneurysmal sac failed to show any communication between the two. There was no thrombosis in the vena cava.

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[6] Brit. Med. Jour., 1891, i, 407; Trans. Path. Soc. London, 1891, xlii, p. 71.

[7] Gaz. Med. di Torino, 1893, xlii, 517.

[8] Edin. Med. Jour., 1985, xl, 896.

[9] Sem. méd., 1900, xx, 151.

[10] Deut. med. Woch., 1904, i, 601.

[11] Bull. et mém. Soc. anat. de Paris, 1903, vi, 463.

The murmur described is simulated by the murmur caused by a rupture between an aneurysm and the pulmonary artery, as proved by cases reported by Clarke[12] and Kaufmann,[13] but in these cases there is not the characteristic cyanosis of the face, head, and chest. Cases are reported of a tumor making gradual pressure on the vena cava in which nearly the same physical signs exist, excepting the murmur. In his case, too, Goldflam[14] says the symptoms came on slowly.

The diagnostic signs, then, of rupture of an aneurysm into the superior cava are: (1) A sudden cyanosis, œdema, and dilatation of the veins of the face, head, neck, arms, and chest. (2) A peculiar, continued, high-pitched murmur, present in about half the cases. (3) The physical signs of an aneurysm of the aorta. A very probable diagnosis of the condition may be made on the first sign alone.

A STUDY OF THE DIFFERENT FORMS OF ALBUMIN OCCURRING IN THE URINE.

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Strictly and correctly speaking the term albuminuria may be limited by some to the excretion of albumins in the urine through the renal tissues; but if the term is considered as applied almost universally it relates to the finding of an albumin in the urine excreted from the bladder. We have accepted the second conception as the basis for this article, which deals with over 5000 examinations of specimens, many of which were sent to the laboratory with the statement that albuminuria was present and with the request to interpret this condition. It has been found that the large majority of medical men recognize but one type of albuminuria—any positive reaction for albumin meaning serum-albuminuria, a view which is erroneous.

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[12] Brit. Med. Journ., 1900, ii, 1701.

[13] Wien. klin. Woch., 1905, xviii, 1004.

[14] Méd. mod., 1892, iii., 416.

Albumin Bodies Commonly Found in Urine. If one studies this subject it is found that albumin bodies of endogenous origin which may occur in urine are numerous, including serum-albumin, serum-globulin, fibrinogen, fibrin, nucleo-albumin, nucleo-proteid, mucin, Bence Jones' albumin ("albumose") and the derivatives of albumin, digestive albumose, and true peptone.

In a recent article[1] we have gone at length into the classification of the albumin bodies which might, theoretically, occur in the urine, the albumin bodies which commonly occur in the urine, and the reasons for releasing from consideration certain of these bodies; we have emphasized four of them which should be borne in mind for common-sense reasons—serum-albumin, serum-globulin, nucleo-albumin, Bence Jones' albumin (albumose); and, furthermore, we have given reasons for adopting the following simple methods of differentiation:

The specimens of urine were subjected to four distinct tests: (*a*) Heat and acetic acid for the presence of an albumin body; (*b*) cold acetic acid with diluted urine to prove the presence of nucleo-albumin or mucin; (*c*) heat and acetic acid, after salting with saturated aqueous sodium chloride solution, thus excluding nucleo-albumin and in many instances bringing out a serum-albumin reaction not detected by the first test, on account of the poor neutral-salt content of the urine; (*d*) the test given for serum-globulin.

Out of 5330 specimens, 4068 (76.5 per cent.) showed reactions for albumin by heat and acid, (*a*) or by salting and heat and acid, (*c*) or by both; and it is upon 3089 of these specimens, in which the albumin bodies were carefully separated, that the percentages of different types are based.

METHODS FOR SEPARATING AND RECOGNIZING ALBUMINS.[2]

(*a*) Test-tube two-thirds full of urine:

Heat upper one-third of urine. Cloudiness may be—serum-albumin, nucleo-albumin, serum-globulin: On addition of 2 to 5 drops of 50 per cent. acetic acid and second heating—increased cloud or lessened cloud persisting.

Albumose (Bence Jones), phosphates, carbonates: On addition of 2 to 5 drops of 50 per cent. acetic acid and second heating—disappearance of cloud or partial clearing.

If the reaction is faint ("faint trace")—probably serum-albumin, nucleo-albumin, or both.

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[1] T. W. Hastings, Albuminuria, Medical Record, July 7, 1906, p. 13.

[2] Hastings, Medical Record, July 7, 1906.

(*b*) The presence of nucleo-albumin is tested for by diluting 3 to 5 c.c. of urine with five to six times its volume of distilled water, adding to one-half of this diluted urine 1 to 2 c.c. of 50 per cent. acetic acid cold, not heating; a diffuse cloudiness in the acidulated one-half is probably nucleo-albumin (may be mucin, but mucin will not respond to the first heat and acetic acid test).

(*c*) Nucleo-albumin is not precipitated by heat and acid in highly "salted" urines, and, therefore, saturated aqueous solution (30 per cent.) of sodium chloride is used to prove the presence of serum-albumin.

To the urine add one-fifth volume of saturated sodium chloride solution, heat the upper one-third, add 2 to 5 drops of 50 per cent. acetic acid, and heat a second time; a persistent cloud is serum-albumin (may be with serum-globulin).

If a massive reaction, there is usually serum-albumin with serum-globulin; may be Bence Jones' albumin ("albumose") or serum-albumin alone or with fibrinogen.

To separate serum-globulin and albumose (Bence Jones' albumin), to the urine add one-fifth volume of saturated sodium chloride solution and 2 to 5 drops of 50 per cent. acetic acid, with or without heating. Cloudiness may be serum-globulin, albumose (Bence Jones' albumin), or, rarely, serum-albumin alone.

Heat the upper one-third. A heavy cloud followed by partial clearing suggests albumose (Bence-Jones). A persisting cloud is serum-globulin or serum-albumin, or both.

Boil the entire tube and filter. A heavy cloud reappearing in the filtrate on cooling is albumose (Bence-Jones' albumin). A faint reaction on cooling, due to deutero-albumose, often appears; this has practically no clinical value.

(*d*) For serum-globulin:

To a test-tube of distilled water add urine drop by drop to 8 to 10 drops, no heating. A cloud (cold) means serum-globulin, which clears after addition of 2 to 5 drops of 50 per cent. acetic acid (cold).

It is sufficient to state that such methods depend upon the properties of the different albumins, whereby they react differently to heat and to acids in solution, free from or possessing a low content of neutral salts, and containing large amounts of neutral salts, properties well known to physiological chemists and not sufficiently noted by the physician. Practical illustration of the results obtained by such methods is seen in these urine examinations in five cases of lobar pneumonia. (See Table I.)

There is only one chance for confusion, that of mucin with nucleo-albumin, since both react in dilute solutions poor in neutral salts to an excess of acetic acid in the cold. Mucin, however, does not react to heat and acetic acid as does nucleo-albumin in urines ordinarily; and when the first test without salting is positive and the second test with salting is negative, the nucleo-albumin is certainly present and may react in dilute solution after the addition of cold acetic acid.

TABLE I.

PNEUMONIA CASES.

	Test *a*.	Test *b*.	Test *c*.	Test *d*.	NaCl per cent.	Amt. c.c.	Sp.gr.	Casts.
CASE I.								
Jan. 24	+	0	+ + +	0	0.68	....	1016	0
25	+	0	+ +	0	0.5	....	1020	H + G
26	+	0	+	0	1.2	400	1018	H + G
27	+	+	+ +	0	0.5	800	1027	H
29	+ + +	0	+ + +	+	0.6	1250	1022	H + G
30	+	0	+ +	0	0.5	800	1025	H + G
31	+	0	+ +	0	0.45	420	1026	H
CASE II.								
Jan. 25	+	0	+ +	+	0.85	1600	1015	H + G
26	+	+	0	0	1.05	2400	1010	0
27	+	0	+	0	0.75	2000	1005	0
29	+	0	+	0	0.75	2450	1006	0
30	+	0	+	0	1.45	1300	1015	0
Feb. 1	0	0	0	0	0.9	2500	1010	0
4	+	0	+	0	0.48	1300	1016	0
CASE III.								
Feb. 15	+ +	+	+ + +	0	0.3	800	1016	H + G
16	+ +	+	+ + +	0	0.2	1000	1016	H + G
17	+	+	+ +	0	0.5	400	1018	H + G
19	+ +	+	+ +	0	...	500	1014	H + G
20	+	+	+ +	0	0.2	600	1014	H
21	+ +	+	+ + +	0	0.3	1080	1014	H + G
CASE IV.								
Jan. 24	+ +	0	+ + +	+	0.63	1380	1018	H + G
26	+	+	+	0	0.85	1000	1016	H + G + E
27	+	+	+ + +	+	0.15	1800	1014	H + G
29	+	0	+ +	0	0.15	750	1012	H
30	+	0	+	0	0.2	1300	1015	H
Feb. 1	+	0	+ +	0	0.25	2400	1016	—
4	+	0	+	0	0.62	700	1020	H
6	+ +	0	+ +	0	0.74	1000	1018	H + G
7	+ +	0	+ +	+	0.8	900	1020	H + G
CASE V.								
Feb. 3	+	0	+	0	0.13	400	1025	0
6	+	+	0	0	0.7	560	1022	0
8	+	+	+ +	0	0.34	800	1026	0
9	+	+ +	+	0	0.34	520	1022	0
10	+	+ +	0	0	0.5	520	1026	0
12	0	0	0	0	0.95	....	1015	0
15	+	+	0	0	0.75	980	1015	0
17	+ +	+ +	+	0	0.55	1180	1014	H
19	+	+	+	0	0.85	2000	1014	H
20	+	+	+	0	0.95	1200	1016	H
24	+	+	0	0	1.00	....	1020	0

+ = faint reaction.
+ + = marked reaction.
+ + + = massive reaction.

H = hyaline casts.
G = granular casts.
E = epithelial casts.

Among the figures following there is no question concerning those for "nucleo-albumin" alone but under those for "nucleo-albumin and serum-albumin" the nucleo-albumin reaction (cold) may have been due in some instances to mucin, and yet we class the reactions as for nucleo-albumin, since mucin is very rarely present in sufficient amount to respond to any test when small amounts of urine are used, excepting in gross infectious conditions usually below the ureters, and in infectious conditions one finds nucleo-albumin as well as mucin, serum-albumin, and possibly serum-globulin. According to Dr. Hoobler's analyses of 1988 specimens, giving one test for albumin, mucin occurred in 349 (17.5 per cent.); but when one uses the heat and acetic acid test mucin does not respond, and it need not, therefore, be considered from a practical standpoint. In three cases only have we been able to isolate mucin and recognize it as such by splitting with mineral acids and obtaining a reducing body from the twenty-four-hour urine output.

As mentioned in the former article referred to, serum-globulin need not be considered apart from serum-albumin, since in these 3089 specimens of albuminuria it occurred in but 5.6 per cent., never alone, and always with serum-albumin; its occurrence was about equally distributed between cases of acute hemorrhagic or parenchymatous nephritis, hematuria, or infection either renal or below the ureters in the bladder or prostate.

Fibrinogen or fibrin occurred in nine specimens from 3 cases, one case of filarial hematuria and two cases of profuse hematuria.

NECESSITY FOR SEPARATING SERUM-ALBUMIN AND NUCLEO-ALBUMIN. The necessity for separating serum-albumin and nucleo-albumin arises when the reaction is faint, since both respond alike to the simpler tests, and serum-albumin has a definite significance, while nucleo-albumin, as far as we know, is of relatively little import, unless possibly indicating the conditions coming under the term "irritation." It is a fact that nucleo-albumin is not commonly present in the urine of chronic nephritis, while it is, as a rule, found in the urine of acute nephritis and subacute nephritis and in inflammatory conditions of the genitourinary tract below the kidney.

In our 3089 urine examinations nucleo-albumin alone was found in 839 (26.5 per cent.) of the specimens. (See Table II.) We have welcomed such positive reaction without serum-albumin in cases of pregnancy and in operative cases in which the kidney to be left behind was thought guilty of a faint albuminuria, particularly, and also in cases of suspected nephritis. With us the commonest occurrence and source of this trace of nucleo-albumin is in the male and from chronic urethral and prostatic conditions. In one case of hydronephrosis with operation its source was undoubtedly renal, and it was the only albumin body present in the fluid from a large non-infected sac, involving a large portion of the renal parenchyma. Whether or not this was a renal nucleo-proteid we were not prepared to prove. In this one case only have we been able to prove absolutely, however, a renal origin for the nucleo-albumin reactions, although it probably not infrequently exists; of the not uncommon prostatic origin we are certain.

The higher percentage of albuminurias in the hospital cases as compared with dispensary cases is striking, and is probably explained by the facts that among the former many were postoperative, and the specimens from nephritic cases were usually examined daily and the non-nephritic weekly.

The discrepancies between the reactions brought out by salting (10, Table II) and between the occurrences of nucleo-albumin (11, Table II) in the hospital and dispensary cases are undoubtedly due to the relatively frequent occurrence of a normal neutral salt content and of nucleo-albumin in the acute and subacute nephritic conditions with infectious diseases and in acute nephritis (hospital cases), and to the relatively infrequent occurrence of a normal neutral-salt content and of nucleo-albumin in chronic nephritis (dispensary cases).

Table II.

	D. Mixed Dispens.		H. Hospital.		D. + H.
	Specimens.	Per cent.	Specimens.	Per cent.	Average per cent.
1. No albuminuria	750	26.5	512	20.5	23.5
2. Total albuminurias	2080	73.5	1988	79.5	76.5
3. Albumin separated.	1101	—	1988	—	(3089.)
Albumin, type not noted.	979	—	0	—	(979.)
4. Nucleo-albumin alone.	269	24.4	570	28.6	26.5
5. Serum-albumin, no nucleo-albumin	344	31.2	176	8.8	20.
6. Serum-albumin + nucleo-albumin	488	44.3	1202	60.4	52.4
7. Albumose (Bence-Jones' albumin)	0	0	0	0	0
8. Serum-albumin + serum-globulin	62	5.6	—	—	
9. Serum-albumin + fibrinogen or fibrin. (3 cases)	9	0.81	0	0	0.81
10. Nucleo-albuminalone or + serum-albumin	757	68.7	1696	85.3	77.
11. Serum-albumin with or without nucleo-albumin	832	75.5	1418	71.3	73.4
12. Reaction after salting only	308	27.9	40	0.21	14.

The necessity for using some such method as salting, heating, and acetic acid (*c*) to bring out or intensify albumin reactions for serum-albumin in urine poor in neutral salts is shown by the fact that 348 specimens (14 per cent.) of the albuminurias reacted distinctly and often markedly after salting only and responded to no other test as well as by the urines from pneumonia cases. Of these specimens many were from 25 nephritic cases, 20 per cent. of the total number of cases of nephritis.

Albuminuria in Cases with Clinical Diagnosis of Nephritis. We do not refer here to those cases of contagious, infectious, metabolic, and toxic conditions which show a disturbance of renal function and probably a transient nephritis, nearly all of which conditions show serum-albuminuria with casts, some of them serum albuminuria without casts when tested with salt solution, heat, and acetic acid, but to cases of well-established Bright's disease in which the first suggestion of the renal condition comes from the symptoms and physical signs.

In proportion to the total number of cases showing albuminuria. 1014, the percentage is small, 12.59 per cent.

As mentioned before, the chronic cases gave no nucleo-albumin-mucin reaction as did the more acute cases. Most important is the fact that many of the chronic interstitial cases, with large amounts of low specific gravity urine, gave no positive tests until the urine had been salted, tested with heat and acetic acid, and allowed to stand for from five to ten minutes.

What we wish to emphasize are the discrepancies between the total cases of serum-albuminuria (1014), the number of cases showing serum-albumin and casts (474), and the number of cases clinically nephritis (125). (See Table III.)

TABLE III.

	Cases.				
	Dispensary.	Per cent.	Hospital.	Per cent.	Per cent.
Albuminuria (=serum-albumin), no casts, clinical diagnosis nephritis	1	0.098	2	0.196	0.29
" " +casts.	104	10.2	21	2.1	12.3
Nephritis=128 cases	105		23		12 59
Albuminuria (=serum-albumin) no casts, clinical diagnosis not nephritis	447	44.	90	8.99	52.9
" " +casts.	174	17.19	175	17.3	34.4
Total cases of albuminuria (=serum-albumin) in 5330 urines	726	71.8	288	28.	100.
Total cases of albuminuria (=serum-albumin). +casts	278	27.4	196	19.3	46.7
No albumin-bodies = clinical diagnosis nephritis	1 exam. 1 visit.		0		0
Nucleo-albumin alone=clinical diagnosis nephritis	0		0		0

Albuminuria does not necessarily, nor does it usually, indicate nephritis, a fact more generally recognized by the genitourinary surgeon than the general practitioner; but it may be indicative of nephritis, and, therefore, cannot be disregarded in diagnosis. From our cases it would seem that albuminuria *with casts* is good evidence of nephritis either temporary, as in the majority of diseases, or permanent in the conditions showing the other evidences (cardiac, circulatory, digestive, cerebral) of Bright's disease.

In no instance has nucleo-albuminuria alone occurred in a case which suggested in any way nephritis.

In only one instance has serum-albumin been absent in a case with the clinical diagnosis of nephritis, in which case but one specimen was examined and the patient was seen but once. In several cases the albuminuria had disappeared entirely after several days or weeks of treatment, during which period the signs and symptoms improved markedly.

Such evidence may suffice for the practitioner, but does not satisfy those of us who look to postmortem evidence as final; for this we refer to the meagre evidence presented by the cases to be published later, which coincide with the clinical evidence.

In only one case and in one specimen was nucleo-albumin found alone with hyaline casts; and in only two cases and two specimens was "no albumin" found with hyaline casts. These observations were made by the same individual. Such occurrences in 5000 specimens of urine do not furnish evidence of value.

We would state, in conclusion, that this material, coming from variedly mixed services—medical, surgical, gynecological, and numerous dispensary departments—gives a fair idea of the average occurrence of albuminuria in practice. We would emphasize the frequency of non-renal albuminuria and the occurrence of nucleo-albuminuria alone in a large percentage of cases; and would recommend the addition of saturated salt solution with subsequent heating and acidifying with acetic acid as the safest test for serum-albumin. In practice, the separation of the albumin bodies is unnecessary.

We extend our thanks to Dr. Thacher, of the Presbyterian Hospital, for placing at our disposal much of the material studied.

THE PATHOLOGY AND TREATMENT OF NEPHRITIS.

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The kidney stands in very intimate relation to all the other organs of the body and has a twofold function of excretion and secretion. Placed as it is, at the point where the metabolic products of the system may be thrown off or held back according to their nature and their properties of being useful or useless in the further activities of the body, the kidney, with unerring accuracy, quickly excretes those substances which have outlived their usefulness in the economy and holds back those which have further value. In other words, this organ, being the osmo-regulator of the blood and, consequently, the regulator of the composition and concentration of this tissue, shows almost intelligent power of excretion.

It is worth while bearing in mind the fact that an exceedingly close relationship exists between blood supply and functional activity, an instance of which is the numerous capillary tufts shoved into the beginning of the tortuous secreting tubules. An additional point of value, however, is that the afferent artery, which breaks up into the capillary tuft, has a greater caliber than the efferent vein leading from the tuft. It is, therefore, easier for blood to reach the glomerulus than to leave it, inasmuch as the pressure in the capillary tuft is greater than in ordinary capillaries owing to the resistance in the narrow efferent vein and its capillary plexus. This double capillary supply, both on entering and on leaving the glomerulus, seems to suggest the idea that the highly organized part of the renal tubules is protected from excessive blood pressure by the interposition of the glomerulus as a block.

I shall not discuss the facts, pro and con, leading to the assumption of our present theory of renal secretion further than to state that the water, salts, and probably the pathological sugar and albumin are excreted by the glomerulus, while urea, uric acid, creatinin, with some water and salts, are excreted by the selective activity of the epithelium of the tubules, certain portions of the tubules having different functions in this regard. Naturally, in organic lesions of the kidney disorders of both secreting centres arise, and we have a resulting change in the composition of the secretion. The rate of secretion, necessarily, rises and falls with the blood pressure and also with the velocity of the current passing through the vessels, being influenced, of course, by the activity of the renal epithelium. From the experimental data at hand we must conclude that the process of secretion in the kidney is one of selective activity and not of pure physical filtration.

Modern Conceptions of Nephritis. Normally albumin does not appear in the urine in sufficiently large amounts to respond to our clinical tests. Its presence, clinically, is said to be indicative of a lesion in the glomerular epithelium, as is indicated by the cases of acute interstitial nephritis without albuminuria described by Councilman and others.

It is a very difficult and even impossible task to draw the line between truly physiological or slightly pathological conditions, and those which are clinically characteristic of nephritis. As Stengel says, "The more accurate urinary examinations and the more careful medical diagnoses of recent years have established the fact that it is necessary to distinguish between nephritis in the sense of the pathologist and nephritis or Bright's disease in the clinical sense." The physiological, cyclical, adolescent, orthostatic, and other varieties of albuminuria are rarely, if ever, accompanied by the urinary findings said to be characteristic of nephritis. Febrile albuminuria, on the other hand, is often accompanied, according to von Leyden, von Leube, Senator, and others, by all the elements found in true renal inflammation. We must, therefore, assume that inflammatory lesions in the pathological sense may be present without causing any lasting results, provided these changes have not seriously hampered the functional activity of the kidney. I quote from Stengel: "The presence of slight intertubular exudation of moderate or even extensive, if uncomplicated, degeneration of the epithelium of the tubules, or of slight alterations in the glomeruli, would not justify a positive assertion that the case was one of Bright's disease in the clinical sense. In all of the cases now in mind, albuminuria, the great criterion of nephritis, is likely to be encountered, and yet the pathological conditions are frequently neither serious at the time nor lasting and progressive in character."

The point of all this is, it seems to me, that we must now consider nephritis simply as a renal manifestation of constitutional or metabolic processes. The clinical picture of nephritis may follow these preliminary disorders, depending on the nature of the irritation of the toxin, the time of its action, and the resistance of the kidney to its action. In other words, nephritis is, in the pathological sense at least, due to a direct irritation of the renal parenchyma by poisons, whether they be exogenous or endogenous in character, which have reached the kidney through the circulation, and which exert their untoward influence on the secreting organ. That direct irritation of the kidneys by bacteria is possible we must grant, although, in this condition, we must assume that the lesions are more focal than when caused by the products of bacterial activity. As Councilman says: "The most easily understood lesions of the kidney are focal in character and due to the presence of bacteria in the tissue. Bacteria often pass through the two capillary systems and lodge in the veins of the pyramids. This is particularly the case in hematogenous infections by Staphylococcus aureus. In this connection comes the question of the passage of bacteria through the intact epithelium. Experimentally this has not been shown to take place. It is true that in certain of the infectious diseases, notably typhoid fever, bacteria are found in the urine, but it has not been shown that the kidneys, in these cases, are intact or that the bacteria come from the kidneys."

In a paper recently read before the Chicago Medical Society, Dr. Brewer, of New York, cited several clinical cases, as well as many original experiments, showing a unilateral multiple abscess formation in the kidney following hematogenous infection by various pathogenic and pyogenic bacteria. Clinically, such cases are manifested by acute gastric symptoms with diffuse abdominal pain, later becoming localized in the flanks, with sudden rise in temperature to 105° F. or more, and accompanied by all of the urinary findings of acute nephritis. Such cases must put us on our guard as to the proper diagnosis of inflammations and infections of the kidneys, inasmuch as the treatment, in the latter cases, seems necessarily surgical. We must remember that not all infectious fevers show nephritis as a common complication, but they may, provided the toxin produced by the metabolic activity of the organism in question, is of sufficient concentration and virulence. Also, in the light of Dr. Brewer's observations, we must consider surgical interference in cases showing the symptom-complex mentioned.

When one considers the etiology of nephritis he finds, in every case, that the causative factor is one which has produced marked metabolic or functional disorders resulting in the production of toxins, irritant poisons, changes in the properties of the blood, and other general pathological alterations, which, in themselves, cause irritative and later inflammatory lesions in the kidneys, resulting in what we call, clinically, nephritis. It must be insisted upon that the nephritis will be of a type distinctly pathological or clinical, depending on factors mentioned above. It may still be nephritis in the pathological sense, without being recognizable clinically.

Owing to this fact, one may not venture a classification of the various types of nephritis without, necessarily, leaning toward the one or the other viewpoint. I shall, therefore, simply treat the subject from the clinical standpoint, leaving out of the consideration the well-established pathological fact that we have types of acute parenchymatous, interstitial, and diffuse nephritis. I fortify my statement that we have the pathological type of acute interstitial nephritis without clinical manifestations, by quoting from Councilman: "The acute interstitial non-suppurative nephritis is found in acute infectious diseases, notably in the acute exanthemata, and consists in the presence in the bloodvessels and interstitial tissue of the kidney of cells belonging to the lymphoid series, which are formed in the bone-marrow, spleen, and lymph nodes, and which are brought to the kidneys by the blood. They accumulate in the renal veins, pass from these into the interstitial tissue, and proliferate actively in both places. Unless the cell accumulation is so great as to interfere with the circulation or nutrition they need not give rise to any interference with function, though usually degenerative changes in the kidney due to the toxins of the disease accompany the interstitial changes. These cells never accumulate in the capillaries of the glomerulus, but are found to a slight extent in the capillaries of the cortex, and chiefly in the veins of the pyramids."

Likewise, in considering the chronic type, care must be taken not to confuse the pathological and the clinical standpoints. The interstitial changes observed in chronic cases are, necessarily, secondary to the parenchymatous if pathology be considered, yet, clinically, we do not find all cases of chronic interstitial nephritis as resultants of the chronic parenchymatous type. We must regard the chronic interstitial type as a phase of a general metabolic disorder manifested through the vascular system.

Influence of Cardiovascular Changes. As is well known, nephritis, depending on its type, may lead to marked changes throughout the cardiovascular system, as well as be the result of such changes. We cannot explain the vascular changes in the same way for the various types. It is self-evident, it seems to me, that the causes leading to a dilatation of the right heart in acute nephritis must be different from those bringing about a hypertrophy of the left heart with secondary dilatation in chronic nephritis.

In the acute forms we must look for our cause in the increased resistance offered through the accumulation of the volume of blood in the vessels. Water, as is well known, is, perhaps, the most difficult substance to excrete in acute nephritis, as evidenced by the marked oliguria or even anuria observed. In consequence of this retention of fluid the right heart must dilate to accommodate the increased amount, and secondarily the left heart hypertrophies in its effort to force the increased load through the system. Depending entirely on how long this increased heart action is kept up without relief, the result will be sooner or later a breaking down of the compensation and we will then have to deal with a case of heart disease. It is to be said that very rarely does such a sequence present itself in acute nephritis, but it may, as many clinical histories will show. Such clinical pictures are best seen in those acute cases with slight œdema and almost complete anuria. The chronic types are much more often accompanied by this sequence of events.

When we consider the chronic forms of nephritis we see that, although the cause may be different in each type, the result is the same, namely, a marked increase in arterial tension. While in the chronic parenchymatous type the retained metabolic products exert an irritative vasoconstricting effect on the vessels, and at the same time stimulate the heart musculature, the vessels in this type become more permeable and œdema results. Later, the left heart, particularly the ventricle, hypertrophies, owing to the twofold cause of stimulation and increased resistance.

In the primary chronic interstitial nephritis the pressure in the arterial system is high and the contraction of the vessels is great, although the amount of urine is increased. However, the urine in this chronic interstitial form is increased only as regards the water and the easily soluble urinary constituents. In the contracted kidney following arteriosclerosis there is marked interference with the circulation in consequence of loss of tone of the vessels, with resulting increased arterial pressure, and in consequence of the injurious effect of the metabolic products of the blood.

The cardiovascular changes in all forms of nephritis may become so marked that they give rise to the symptoms requiring our greatest efforts, the kidney lesion then becoming of secondary importance from the therapeutic standpoint.

Albuminuria and Cylindruria. As will be granted from what has preceded, albuminuria is a very common finding in conditions which are of general nature and which give rise to nephritis of the pathological rather than of the clinical form. It is an every-day occurrence to find albuminuria accompanying febrile conditions, yet no signs of clinical nephritis appear to settle the diagnosis. It may be stated as a dogma that albuminuria, *per se*, is not necessarily indicative of nephritis in the clinical sense. Yet, judging from our knowledge of pathology, some lesion must be present in the kidney to permit of such a condition. It is quite possible that the so-called alimentary albuminuria is accompanied by some irritative, though slight, lesion in the kidney, resulting in the removal of the albuminous substances from the blood, yet such substances are, to all intents and purposes, foreign to the blood and may simply be excreted as such.

Albumin is usually considered as excreted through the glomeruli, but, in addition, it may pass from the interstitial bloodvessels and lymphvessels into the urinary tubules, so that, primarily, we may conceive of a glomerulitis whenever albuminuria exists. Either disturbances of circulation, irritation of the parenchyma of the kidney, or changes in the blood may be held responsible for the pathological albuminuria. Space will not permit of a discussion of the theories of Semmola, Lepine, and Rosenbach. It seems to me that it is not improbable that we may, by diligent research, show that differences exist in the albuminuria from various causes. I mean by this that we may find, and certain evidence from experimental work seems to strengthen the assumption, that the albumin excreted in pneumonia, for instance, is different chemically from that observed in nephritis following exposure or excesses. Whether this is or is not the case will be shown, I believe, by the application of the principles so well worked out by Emil Fischer. It is not inconceivable that the various etiological factors of nephritis, particularly the toxic products of bacterial growth, may, in their formation, cause the disintegration and resynthesis of the serum albumin into proteid bodies with different arrangement and different concentration of the various amino-acids, of which they so largely consist. Such bodies would give our clinical tests for albumin and would not be distinguishable from serum-albumin from the clinical point of view.

Regarding cylindruria, it is to be said that here, also, we must be on our guard lest we err in our diagnosis of nephritis. Casts of all kinds appear in the urine in cases showing no clinical manifestations of nephritis, yet we must assume, if our ideas of the origin of casts are correct, that a lesion of the kidney exists. As Emerson says, "Casts alone are no index of the anatomical renal condition. Their most brilliant display is in non-nephritic conditions; the most serious conditions in nephritis may be accompanied by few or none. The more normal the cells the better do they seem to form casts when disturbed: in chronic conditions the cells seem to become accustomed to their condition and to form few casts or none."

Chloride Retention. Since the publication of Widal and Javal in 1903 a large number of French and a few German workers have busied themselves with showing that sodium chloride is, contrary to our well-established ideas, a decidedly harmful substance in cases of nephritis. Although Widal and Javal, Achard, Loeper, Claude, Bohne, Castaigne, and others did not originally apply their idea to any but the parenchymatous variety of nephritis, later workers, such as Achard and Paisseau, Brandenstein, Calabrese, Chauffard, Olmer and Audibert, Merklen, Lesne, Vaquez and Laubry, Strauss, and others have extended this original idea to interstitial nephritis, cardiac and hepatic dropsy, epilepsy, etc. These workers have found that the chloride is retained in the system, that concurrently the œdema is increased, and a greater albuminuria is noted. Some recent work of Miller has shown that there is quite as much retention in normal individuals as in nephritics. That œdema is not observed in the normal goes without saying. Ferrannini has shown that although chlorides are retained in nephritis and albuminuria is increased, the general betterment of the condition warrants the use of this substance as a subcutaneous injection. Rumpf, in a recent article, shows that the idea of retention of sodium chloride as an etiological factor in the causation of the œdema of nephritis, is not well grounded. Richter confirms this and argues that sodium chloride is no more responsible for the œdema than are many other factors, especially the amount of water intake. Marischler shows that the kidney of parenchymatous nephritis is permeable for sodium chloride and that the retention is explained by the primary retention of water. Kövesi and Roth-Schulz confirm this idea, and go farther into the physical explanation of the œdema. We observe, therefore, that this assumption of the causal relation of retention of chlorides to the œdema is debatable.

It may be well to ask, How does sodium chloride act in the normal and how in the nephritic metabolism? When NaCl is administered by the mouth it, of course, is absorbed from the stomach and bowel. The blood becomes more concentrated; hence, water passes from the tissues into the blood making this hydremic. The excess of water and of salt is normally excreted by the kidney, since this organ is the osmo-regulator of the blood. NaCl acts, therefore, just as any other salt, such as the citrate or acetate would act, as a diuretic, owing simply to its "salt action." The question might be asked in this connection, Why normal individuals show a retention of chloride? The answer must be rather vague in the present state of our knowledge. The amount of NaCl in the blood, according to Hammarsten, is 6 per 1000, and this amount remains almost constant. With food containing an excess of NaCl, this excess is quickly eliminated, and with food poor in chlorides the amount in the blood first decreases but later increases, after taking chlorides from the tissues. The secretion by the urine is thereby diminished and apparent retention occurs. This retention, it must be insisted on, is only apparent and vanishes as soon as the proper osmotic equilibrium has been established between tissues and blood. According to Marie there are two stages in the retention of chlorides, and these stages depend, of course, on the laws of osmotic pressure and of chemical equilibrium. The first stage of retention, as Marie puts it, is that of fixed chloride (tissue), namely, the combination of the NaCl with the proteids of tissues, which assumption has been experimentally shown to be possible by van Bemmelen, but is doubted by Bugarsky and Liebermann. The second stage is that of circulating (interstitial) chloride. This added point shows us how in the first stage we get a retention, if the NaCl of the blood is insufficient, owing to a poor intake, and is then increased by food rich in NaCl.

If now the kidney is "insufficient," or, in other words, is unable, owing to the inflammatory processes affecting its excretory activity, to excrete the excess fluid and its constituents, there occurs a retention of salt in the circulatory system, a hydremia, and at the same time a plethora, vascular pressure is increased, transudation through the capillaries follows, an attempt is made to adjust the osmotic pressure of the blood and the tissue fluids, salts pass into the tissues, which, in time, become hypertonic, and, in consequence, water follows the salts into the tissues, and œdema occurs as a combined result of increased vascular pressure with transudation and the retention of salts in the tissues. This theory of œdema as a result of osmotic exchange between the blood and tissues was advanced by Loeb in 1898, and has been reached by the French school, especially by Widal and Javal, working from different standpoints.

It is difficult to say just why nephritics show increased albuminuria after increased intake of NaCl, yet one may call to mind the results of Hoppe-Seyler, von Wittich, and Nasse, showing that an albuminous solution filters stronger the greater its content of salt. This purely chemical fact may also be applicable, as far as the purely physical theory of filtration through the kidney is concerned. On the other hand, one may assume that the kidney has become more permeable for albumin, or that the albumin itself has become more diffusible, inasmuch as various workers have shown that normal individuals, in time, show an albuminuria under the influence of increased intake of NaCl. There are difficulties in the way of both explanations. In the first place, if the kidney has become permeable for albumin, then would it not be permeable, at the same time, for the increased chlorides? If the albumin is more diffusible, it is not as diffusible as the NaCl, and yet the latter remains and the former passes out. We must again revert to the osmotic theory for our explanation. Proteids possess only a slight osmotic value, while the salts have very large equivalents. The osmotic and the diffusion currents extract the fluid and the salts first from the tissues and then later from the blood back into the tissues. The proteids are not affected by this interchange, unless we may assume, with Semmola, that some change in the constitution of the proteids, by which they become more diffusible, is brought about in nephritis.

In this connection, I wish to mention some experiments which I began under Senator and have continued at the Cook County Hospital in Chicago. It was found that the urine of nephritics showed the separation of a coagulatory inhibitory substance and at the same time of a distinct ferment with marked peptic characteristics. This latter was in all probability simply an increased amount of the normal peptic ferment found in the urine by Grober, Gehrig, Grützner, Mathes, Rosenberg, Stadelmann, and others. This separation of a substance in the urine which favored or inhibited coagulation of proteid in pneumonia has been previously worked out by Lenobel, Kun, and Lochbihler. It is possible that the albumin becomes more diffusible solely through the presence of these two substances whose identity I am at present unable to disclose. It is, however, a very strange fact that NaCl will increase or cause an albuminuria. The explanation best suited, it seems to me, is that we have a pure toxic albuminuria. This toxic action of NaCl is well known physiologically from the work of Loeb, Moore, Neilson, and others.

Œdema. I shall refer in this connection only to the œdema caused by the lesions of the kidney resulting in certain pathological changes of the metabolism of the fluids and solids of the blood and tissues. I exclude a discussion of cardiac and hepatic œdema as well as of "hydrops cachecticus," which have no direct place here, although I am convinced of the correctness of the reasoning of Kövesi and Roth-Schulz that the cause of cardiac and renal œdema is a common one.

We must abandon the old conception of Bright's that œdema is caused by hydremia of the blood resulting from the loss of albumin, in consequence of which the watery constituents more easily passed out of the vessels, just as we must give up Bartels' and Stewart's ideas of a hydremic plethora, because we can show that retention of urine, *per se*, will not bring about an œdema. Cohnheim assumes an inflammatory change in the vessels by which they become abnormally permeable. Senator remarks: "Under the influence of certain injurious properties of the poison circulating in the blood, the capillaries of the glomerulus suffer first and then, if the injurious action be strong enough and long enough continued, the interstitial and other vascular areas outside of the kidney, such as those of the skin and serous cavities, become affected. A glomerular affection is always to be looked for when œdema is present, but not vice versa."

Kövesi and Roth-Schulz would make the insufficiency of the kidney in itself responsible for the changes resulting in œdema. The changes in the kidney excite abnormal metabolic processes by increasing the watery and solid constituents of the blood and preventing the elimination of the poisonous toxic and autolytic products. As a result of increased vascular pressure combined with abnormal permeability of the capillary walls and increased osmotic change œdema follows. It is well to remember in this connection the role played by the perspiration, both sensible and insensible. As Bidder and Schmidt, Seegen, and others have shown, the amount of perspiration depends on the temperature and the degree of saturation of the surrounding air. However, it is a well-known fact that nephritics perspire very slightly. von Koryani comes forward with an explanation of increased osmotic pressure of the blood, raising the point of evaporation of fluid, and, hence, decreasing the perspiration. It is possible thus to explain a part of the effect of NaCl in increasing the œdema by lessening the loss of water by the skin and lungs. The amount of fluid taken in is also a large factor in the causation of the œdema. As the fluid is absorbed just as it is normally, the hydremic condition is increased, and with the resulting insufficiency of the kidney, water elimination being the first abnormal phase noticed when the kidney becomes affected, aggravation of an œdematous condition follows. There is thus a distinct reason for limiting the intake rather than increasing it, as we cannot flush the kidney in this way owing to its lessened excretory activity. The result is an overburdening of an already highly taxed heart and an increase of the œdema.

Uremia. In this discussion, up to this point, I have not attempted to take up the effects of nephritis on the system in general. Under the head of uremia one must bring in certain points of general pathology and pathological chemistry in an attempt to show that this condition is a result of purely metabolic processes. As œdema is a combined result of renal and vascular causes on the metabolism, so is uremia the result of changes on the cerebral centres and on the digestive system, due to disturbances in general metabolism, together with insufficient kidneys. The subject of autointoxication is one of general interest whether it be enteric, hepatic, or renal.

The theories advanced as to the causation of uremia have for the most part as a basis the idea of overloading of the blood with urinary constituents as a result of insufficient kidneys. Opposed to these theories are those that hold that hydremia with resulting œdema of the brain is at the bottom of the symptomatology. These latter ideas are especially advocated by Rees and Traube, but do not seem to be convincing, inasmuch as certain factors assumed by the advocates are at times missing and hence cannot be regarded as causative of all cases. I refer here, of course, to the hypertrophy of the heart and to the brain œdema. We must, however, grant that the œdema, when present, may be responsible for certain nuclear symptoms observed in uremic attacks.

Space will not permit an enumeration of all the factors advanced in support of the chemical theory as opposed to the physical. We all know that the retention of almost every constituent of the urine has, at one time or another, been held to be as the causative factor in uremia. One by one these substances have been abandoned until now we cannot grant that any one substance is specific in the causation of this group of symptoms. From the time that retention of urea as an etiological unit was advanced by Wilson until the list had been exhausted by Bouchard in his assumption of organic urotoxins, we have been obliged to change our ideas of the factors responsible for the trouble under discussion. Opposed to these views are those of Frerichs, Demjankow, and the closely related one of Treitz, which assume an ammoniemia as the causative agent.

Strauss found that ammonia was increased in the blood, but at the same time other nitrogenous bodies (neither albumin nor urea) are present, which must play a role in the etiology of uremia. He shows that while the salt content of the serum in uremia does not change, the amount of the nitrogenous constituents contained in the hydremic serum, that is, the retention nitrogen, is, as a rule, increased to a great extent. In connection with this work of Strauss one must mention the findings of Gumlich and Richter that ammonia, alloxur bodies, and extractives are markedly increased in the urine of uremics. It is unnecessary for me to discuss the relation of an increased ammonia excretion to an increased acid formation in the tissues. This regulation of the metabolism is a well-established fact. This taken with the idea of Strauss gives us a very rational basis for the etiology of uremia. Recently, Croftan, reviewing the facts given above, as well as certain points brought out in his own experience, advances the theory that uremia is primarily dependent on insufficiency of the liver, following a general metabolic disorder, and that the kidney is only secondarily important in the causation. This theory has a certain element of plausibility, owing to the fact that, in just such cases of insufficient liver, disturbances of the urea formation, ammoniemia, and irregular autolysis occur and lead to increased acid formation either of the amino or oxy series. However, were it not for the disturbance of the excretion by the kidney, uremia would not occur. The general metabolism of the proteids is at fault in the theories advocated by Senator and by Croftan. As Croftan says, "The liver is not the only organ affected, and uremia interpreted as above is not so much the result of hepatic derangement as of a general metabolic disturbance."

This view of the terminal syndrome of some forms of nephritis is quite in harmony with our idea of nephritis as primarily a constitutional disorder with secondary involvement of the kidneys.

TREATMENT. In a discussion of the treatment of the various forms of nephritis emphasis must be laid, at the beginning, on the individual factor in the handling of our patients. It is very irrational to order, for instance, an exclusive diet of milk in all cases, just as it is unsound treatment to deprive all of the nephritics of dark meats. Each case is a law unto itself, and, in consequence, I cannot attempt to do more than lay down general principles which may be varied to suit the particular case in hand.

Dietetics. No factor in our handling of a nephritic patient plays a greater role than the dietetic treatment. A rational basis for such treatment has at last been worked out, thanks to von Noorden and others, so that we are today working with surer principles than before.

As a general rule, it may be said that, in the beginning and during the height of an acute nephritis, starvation for four or five days is the best treatment. Of course, this starvation diet need not be absolute in all cases, as for instance in the lighter types which we observe in most fevers; but in the forms following scarlatina, for instance, starvation more or less absolute depending on the intensity of the process, as shown by oliguria and marked œdema, must be practised. It may be stated here that we should not, for reasons previously mentioned, judge too severely from the amount of albumin or the number of casts; but we should rather watch the clinical symptoms closely for signs of general disorder. It is much better practice to follow the œdema and the heart symptoms rather than the urinary in drawing conclusions as to the severity of a special case. As we know, recovery usually follows in cases of acute nephritis if a certain period of danger can be passed through. It is our duty to avert this danger, if possible, and, therefore, starvation is to be forced in cases of greatest severity, limiting the intake of fluid as far as possible, and giving ice to quench thirst.

It is a matter of common knowledge that in acute nephritis water is the substance most difficult to get rid of through the kidneys. How irrational then is the idea of ordering a rigid milk diet and forcing water. The main effect of such treatment will be the overloading of the blood with material that cannot be excreted, and thus increasing an already existing œdema, and, at the same time, causing the heart to work harder to force the increased volume of blood and, in consequence, increasing the danger of hypertrophy and later dilatation of this organ.

In cases which are less severe and are not accompanied by a high degree of oliguria and œdema, milk to which has been added a little calcium carbonate to form the insoluble calcium phosphate may be given in quantities not to exceed, on the average, three pints, and the remaining caloric value necessary to maintain the standards of the system may be made up of fat in the form of cream and cereals. It must be remembered that, in our efforts to protect the kidney from overwork, we must not go so far as to injure other organs and, at the same time, the whole organism. The principle of "Schonungstherapie" has certain natural boundaries and these must not be overstepped. Our idea must be to preserve the general nutrition at as high a standard as is compatible with the best working powers of the kidney. We therefore gauge our diet largely by the degree of oliguria and œdema, decreasing the former as the latter increases, and vice versa. Meat broths should be interdicted to the last, inasmuch as these contain, practically, nothing but uric acid, purin bases, and creatinin, and, in consequence, can have very little nutritive value and may irritate the kidney attempting to excrete them.

In the later stages of acute nephritis the food may be gradually increased as desired, care being taken to avoid a large excess of proteid. As is often the case, three or four quarts of milk are given daily in the convalescent stage. Such treatment is bad because the fluid, finding its way into the circulation, may overburden a weakened heart; furthermore, the amount of proteid thus introduced is unnecessarily high. It may be said that it is unnecessary to limit the patient to white meats by depriving him of the red meats. No difference is found between them as to their influence on the general metabolism and on the regenerative processes in the kidney.

Exactly the same procedure as outlined above should be followed in cases showing acute exacerbations of the chronic form.

When we consider the chronic types of nephritis we have a different viewpoint to study. These types are essentially chronic, that is, they continue for long periods of time. As curing such a case is, at present, out of the question we should direct our efforts to maintaining the general body activity at as high a plane as is consistent with the condition of the kidney. Sooner or later the chronic forms, particularly the interstitial type, become practically heart cases and should be treated as such. We are, therefore, justified in allowing the patient more or less latitude in the choice of his diet, remembering certain precautions. The intake of fluid should be limited to some extent. The idea of forcing a milk diet with plenty of water is harmful not only to the kidney, but, what is more important, to the heart. A liberal proteid diet is permissible and no difference need be made as to white or red meats. Condiments are to be avoided as far as possible. Fresh fruits and vegetables may be ordered as desired, recalling the fact that von Noorden advises against the use of radishes and celery, but permits asparagus. Coffee, tea, alcohol, and tobacco should be used with great moderation.

In regard to dechloruration, it is to be said that the limitation of salt in the diet is unnecessary, since it seems to me that the theory upon which its deprivation is based is not tenable. Experiments are still necessary to prove that the œdema is dependent on the retention of salt and also to prove that the salt is actually instead of apparently retained. It is true, however, that some cases are benefited by dechloruration, but it seems to me doubtful that such results are due entirely to the withdrawal of salt.

It must be recalled that each person needs a certain definite amount of energy to maintain himself in healthy equilibrium. With a nephritic, the same thing is true within certain limits. As the energy comes from the food it is rational to construct the diet with definite caloric values in view, remembering that it is advisable to restrict the fluids to some extent and to confine the proteid intake within 100 grams (on a basis of 70 kilograms of body weight). If the reasoning of Chittenden and Folin is correct we must reconstruct our ideas of the proteid need of the system to a basis far below that of the old-established formula of Voit (118 grams of proteid on a basis of 70 kilograms of weight). An excess of proteid is the dangerous thing in the dietary of a chronic nephritic.

We must not forget in our dietetic treatment of nephritis that the digestive powers of the patient should be carefully watched. The digestive tract is a prolific source of toxemia and should, therefore, be carefully regulated. Likewise, the functional power of the liver should be maintained at a high standard to protect the system against accumulation of abnormal products of a deranged hepatic metabolism.

Hygienic and Physical Therapy. Almost as much importance attaches to these phases of the treatment of nephritis as to the dietetic. It is naturally in the chronic types that we have to do with such measures to the greatest extent. Of course, in the acute forms we resort to sweating and purging processes with the idea of relieving the kidney of unnecessary work and at the same time of controlling the œdema. As these sweating processes are, in some cases, very weakening, it is good practice to stop them at the first sign of weakness of the heart, provided no uremic symptoms are present; if these develop, we must, of course, use all our efforts to control this condition. It is irrational to allow the free use of fluids and then force the sweating process, since both treatments are followed by weakness of the heart.

In chronic types, hydrotherapy plays a helpful role in aiding the elimination of water and salts, but it in no way aids the excretion of the harmful nitrogenous products. Proper clothing, preferably woollen, and bathing are to be ordered, and the patient should be directed to spend as much time as possible in the open air. The drier the air the better, as this aids the evaporation from the skin. Exercise is a very important element in the proper handling of these patients, but should never be indulged in to excess. Exercise tends to strengthen a weakened heart, but if excessive will lead to serious complications, as the heart is necessarily under great strain in all cases of chronic nephritis. Moderation in all things is the keynote of the hygienic and physical treatment of these cases.

Symptomatic Treatment. There are several indications for the treatment of nephritis which may fall under this heading: (1) Maintenance of cardiovascular equilibrium; (2) regulation of the œdema; and (3) regulation of the bowels and of the liver. The first two of these are more or less closely related, inasmuch as the œdema is partially a result of disturbed cardiovascular equilibrium.

The maintenance of the cardiovascular equilibrium is, in most cases, of the greatest importance. As the essential feature of the circulatory change is a high arterial tension with or without œdema, it is readily seen why we should be on our guard in following such cases. The œdema may come from the kidney lesion direct or later from the breaking down of a compensated heart. It is usually only a matter of time until this latter factor enters into the case. It is important to recognize which of these two factors is predominant in the causation of the œdema, as the treatment is somewhat different in each case. In those cases in which the heart is fully compensated, our indication is to regulate the blood pressure within safe bounds, a certain degree of increased pressure being necessary. These cases should always be followed with some instrument to indicate the pressure, as a routine. As stated above, a careful dietetic and hygienic regulation, together with free action of the skin and bowels, is usually adequate in controlling the arterial tension and œdema. If these measures do not prove effective, we must use some vasodilator drug or a combination of such drugs in our efforts to maintain a proper equilibrium. The nitrites of sodium or potassium are often found beneficial. Erythrol tetranitrate or nitroglycerin is often given with this object in view. As a general rule, we should resort to vasodilatation whenever the pressure in the adult male reaches 150 mm. of mercury, and at even lower pressures in the female. Diuretics are unnecessary and do no good in purely renal œdema. We may resort to colonic irrigation, which may prove useful in reducing the arterial tension and œdema.

There is no indication for digitalis in the treatment of nephritis as long as the heart is compensated. However, when signs of failing compensation appear and the œdema becomes one of the cardiorenal type, digitalis is the drug to use. As long as the preparation is trustworthy any form of this drug may be used, although we are taught that the infusion has special merit in such cases. Care should be taken to watch the action of the drug lest its action becomes cumulative and the second and third stages of its action become manifest.

I have spoken already concerning the proper regulation of the bowel and the liver. I need only say here that this indication is very important, since we have in the digestive tract, as well as in the liver, a source of constant danger. Enteric and hepatic toxemias are too common to need discussion.

Tension of the skin from the œdema is to be relieved by puncture under aseptic precautions. Effusions into the various serous cavities are to be handled by puncture or paracentesis, depending on the location.

As the heart becomes more and more weakened, we find that the patient complains of dyspnœa, dizziness, etc., in the reclining posture. In such cases the upright position is best suited to ease these symptoms.

Care should be taken to watch for symptoms of uremia and to give brisk purgatives and diaphoretics as soon as precursors, such as stubborn headache, dizziness, nausea, vomiting, etc., are noted. In view of recent advances it might be advisable to force an alkaline treatment with the view of neutralizing the excessive acids produced. Venesection may be resorted to if the blood pressure gets too high. Subcutaneous and even intravenous saline injections will often aid the system in throwing off the substances which are at the bottom of the trouble

Surgical Treatment. In recent years surgical treatment has been advocated, especially by Edebohls and Ferguson, in the chronic types of nephritis. Briefly stated, the surgical intervention consists in nephrotomy or decapsulation. This procedure leads to an increased elimination by drainage of the accumulated products. As the operation is done in the primary stage of anesthesia, the untoward effect of the anesthetic is very much lessened. The whole operation consumes only three to four minutes, and, judging from case reports, is often very beneficial. Much may be expected of this form of treatment, as it is surely rational in some cases and is successful in many.

In cases such as those described by Brewer, nephrectomy seems to have yielded cures. It may be that, in these forms also, decapsulation with drainage will be useful; it will lessen the danger of operative procedure.

THE USE OF THE X-RAYS IN UNRESOLVED PNEUMONIA.

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That exposure to the x-rays might be useful in the treatment of unresolved pneumonia was suggested by one of us[1] in discussing the effects of the x-rays on metabolism in leukemia. It was then stated that the remarkable effect that this agent usually exerts upon metabolism is most readily and most satisfactorily explained by considering it to be due to action upon ferment processes normally resident in the tissues; the action in this instance apparently being to accelerate autolytic processes inherent in the tissues. The reasons then evident for adhering to this view were given at that time, and continued studies of the effect of x-rays upon metabolism in various diseases—studies now amounting to twelve—have emphasized our opinion that this is the most satisfactory conclusion to be reached. Further consideration will be given this point in a more extended discussion of the accumulated results in the various conditions studied, and it is, therefore, unnecessary to burden the present communication with it.

Granting the correctness of the view mentioned concerning the action of the x-rays, the idea at once presents itself that treatment with this agent may be beneficial in many conditions in which metabolism is not sufficiently active; but there is great difficulty in determining this point, on account of our very limited knowledge of most conditions in which such metabolic inactivity is suspected of being present. As was said, however, in the paper first referred to, our present knowledge of the process of resolution of a pneumonic exudate indicates that unresolved pneumonia is the most evidently suitable condition in which to try the effect of the x-rays from this point of view. We know that ferment processes cause the digestion and resolution of the exudate in the ordinary pneumonias that pursue a satisfactory course; and lack of resolution, while sometimes really a continued inflammation, appears from Flexner's observations to be often due to insufficiency or absence of digestion of the exudate. In the latter case evidently the rational therapy is, if possible, to excite or accelerate the action of the ferments that should digest the exudate.

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[1] Musser and Edsall. Trans. Assoc. Amer. Phys., 1905; Univ. Penn. Bull., September, 1905.

The possible suitability of the x-rays as a therapeutic measure in this condition led to its being used, in April, 1905, in a case that has already been mentioned.[2] Since that time we have kept under careful observation the cases of delayed resolution of pneumonic exudate that have come to our notice, with a view to choosing from them, for this treatment and for a study of metabolism while under treatment, those cases that seemed fairly certain not to undergo resolution under ordinary treatment, or to resolve so slowly and imperfectly as to lead to permanent crippling of the lung. We limited our choice also to cases in which noteworthy fever and all other evident signs of continued active pneumonitis were absent.

Of the five such cases that became available—all fever and evidences of distinct toxemia having disappeared, and the signs of solidification having been stationary for at least ten days or a fortnight—three patients grew weary of the waiting that we had imposed upon them and insisted upon leaving the hospitals in which they had been kept under observation, so that our further studies of this question have been confined to two cases. The conditions of metabolism observed in these cases while under treatment with x-rays were, however, so striking and the clinical effects in them and in the case previously mentioned appeared so uniformly good, that it seems proper to describe the results in them, in order to encourage a more extensive test of the treatment. In such tests, however, a few considerations need to be kept in mind, in order that the conclusions may be just.

In the first place no results can be expected unless the duration of the condition has been reasonably short—a few weeks at most—

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[2] Musser and Edsall, loc. cit.

for, if organization of the exudate has occurred, a satisfactory result of treatment is almost inconceivable.

Secondly, the condition should be chiefly a real lack of resolution and not a continued inflammation of the lung. We are unable to state what effect the x-rays may have upon chronic pneumonia, but we are not dealing with this point; and in testing the effect upon delayed resolution as such, active continued inflammation should be excluded as far as possible. To exclude even slight grades of chronic inflammation would be exceedingly difficult, for such slight grades produce no symptoms that are distinguishable from those due to the exudate itself; and, indeed, a low-grade inflammation is probably always present, even comparatively early, and certainly when organization begins. Such a condition we could not reasonably desire to exclude, but we should exclude those cases in which decided fever and other evident symptoms of persistent active inflammation and toxemia exist; for, when these are the chief abnormalities present, resolution of the exudate may be impossible, and may also be of little or no value for the time being. In the low-grade inflammation, however, that appears to be due largely, and perhaps entirely, to the persistence of the exudate, resolution of the latter would probably be accompanied by cessation of the inflammation.

Thirdly, it is excedingly important to determine, as far as possible, that one is actually dealing with unresolved pneumonia and not with tuberculosis. Many cases that are hastily thought to be unresolved pneumonia are, of course, unquestionably tuberculous; and judgment as to the value of a treatment of unresolved pneumonia cannot be based upon them. If cases fail to resolve under the influence of the x-rays, it should be determined before using them, in reaching a decision, whether they are or are not instances of tuberculosis.

Finally, we would earnestly recommend that the use of the x-rays in the treatment of cases of this or any other kind be undertaken with a proper realization of the fact that there are dangers associated with it; and the treatment should, therefore, be begun tentatively, and with brief exposures and small doses, until it is determined in the individual case that serious results are not likely to ensue. This is an additional reason for excluding those cases in which there is marked fever and in which other evidences of toxemia are present. For reasons that we shall point out in another paper, we are convinced that persons that are already the subjects of a noteworthy degree of toxemia are more likely than others to be dangerously affected by exposures to the x-rays; and until our knowledge of the contraindications to the use of the x-rays becomes better defined, we believe that with all such persons the use of this agent should be withheld unless there are urgent reasons for administering it.

We would also at this point, and for the reason just given advise that the x-rays be not tried, at least until their effects are better understood, in the treatment of pneumonia during the course of this disease. Such treatment has already been used purely empirically, and if we are correct in thinking that resolution may be hastened in unresolved pneumonia by the use of the x-rays, a very natural corollary of this idea would be the thought that exposures to the x-rays during the course of pneumonia might hasten resolution and shorten or favorably modify the course of the disease. We do not, however, subscribe freely to such a notion, the chief objection to it being that the most important factor in the course of most pneumonias is not the presence of a mass of exudate in the lung, but the presence of a dangerous toxemia. Resolution of the exudate might, if it could be accomplished, be associated with control and reduction of the toxemia, but it is equally possible that it might not influence the toxemia to an important degree; and the point we would make at present is that whatever might be the result upon the local mass, there is actually serious danger that, if resolution could be accomplished, the sudden digestion of the exudate and absorption of its products might, if occurring during the course of a severe toxemia, be productive of much more harm than good.

The main clinical features of our cases will first be given, including the case that has already been briefly reported in the paper referred to.

Case I.—A man, aged fifty-six years, was admitted to the Episcopal Hospital in the service of Dr. A. A. Stevens, on March 13, 1905. He had been taken acutely and severely ill three days before with high fever, dyspnea, and pain in the right side; and presented the usual signs of a pneumonia involving the whole right upper lobe. His disease ran a moderately severe course, and crisis occurred on March 20. The fever and the signs of toxemia disappeared at this time. but the consolidation persisted, and was still present and but little changed when seen by Dr. Edsall on April 1. There was no noteworthy change, except a very moderate degree of lessening in the intensity of the signs, up to April 21, on which date it was ordered that he be given over the affected area five-minute exposures to the x-rays. These were continued for four days.

During Dr. Edsall's temporary absence from town, Dr. George W. Norris kindly watched the course of the case during the next week. There was no special change in the man's general condition, which was already satisfactory, but on April 23 it was noted that there were many rales in the affected area of the lung and that the breathing was somewhat cavernous in type in the upper portion of the lobe, below this bronchovesicular. Three days later he was noted still to have a moderate number of rales in this lobe, while the breathing had become bronchovesicular everywhere. The dulness had greatly decreased. When seen by Dr. Edsall on April

27 the rales had disappeared; there was very slight dulness over the upper half of the lobe, with no dulness elsewhere; the breathing was a little weak over the whole lobe, expiration being slightly prolonged. A week later, when discharged, there was, except for slight weakness of the breath sounds over this lobe, practically no abnormality left. The man's general condition was entirely satisfactory.

Case II.—A Norwegian sailor, aged twenty-eight years, was admitted as a British Consul case to the University Hospital under the care of Dr. Hollingsworth Siter, who kindly gave us the opportunity to study the patient. It was difficult to secure a detailed history from him; but it appeared that while at sea he had been taken acutely ill with fever, dyspnœa, and pain in the left side. He had been ill for eight days, when he had had an apparent crisis; and the day following, when he arrived in port, he had been sent at once to the University Hospital. When admitted his temperature and pulse were normal and his general condition was entirely good, but he still had the signs of marked solidification in the left lower lobe, involving the whole of the upper part of the lobe and reaching down to the level of the ninth rib.

For three days there was no change in these signs; but throughout the next five days there appeared to be a slight improvement, the dulness becoming less intense and the breath sounds less harshly bronchial. From this time on, however, for two weeks the signs remained stationary; and when he was first exposed to the *x*-rays he had, over the whole upper part of the left lobe down to the level of the ninth rib, a moderate degree of dulness, with moderate bronchial breathing and increased fremitus. Rales were absent. An *x*-ray picture was taken at the first exposure, and this showed a moderately intense shadow in the area corresponding to the signs described.

He was exposed daily for seven days. Two days after the first exposure there was no striking change, although it was noted that the dulness was perhaps less marked and that there were a few fine rales in the affected area. Two days after this it was noticed that the dulness had receded upward for about an inch and a half and that even above this point the dulness was less marked; the breathing was much less harsh, and was bronchovesicular; it could not be determined that the fremitus was increased, and there were many fine rales over the upper part of the lobe. Three days later, at the end of the experiment, no abnormalities could be discovered, except slightly weak breathing over the diseased lobe and a small area of dulness, about an inch in diameter, at the upper extremity of the lobe. An *x*-ray picture taken at this time showed nothing abnormal except a small, indistinct shadow at the area in which a trace of dulness was still found. The man appeared to be entirely well in other ways.

Case III.—A Cuban, aged thirty-four years, was admitted to the Pennsylvania Hospital, in the service of Dr. Arthur V. Meigs, who kindly gave us the opportunity to study him. Dr. Longcope also made frequent notes of his case, of which we shall give an abstract, and was likewise kind enough to watch carefully over the details of the weighing of his food in the metabolism experiment, the securing of the twenty-four hours' urine, etc.

The patient was admitted on the fourth day of an illness that had come on with the usual symptoms of an acute pneumonia of the right side. Examination of the right lung showed dulness over the whole lower lobe, with bronchial breathing and increased fremitus and vocal resonance, and with fine crepitant rales over this area. The upper portion of this lung was negative, as was the left lung. The examination, otherwise, is of no consequence. On the 5th, his condition was worse, the pneumonia having extended throughout the lung. The signs over the lower lobe, however, had decreased somewhat in intensity. On the 8th, he had the usual signs of crisis, but the lung remained solidified. Three days later the lower lobe appeared to be slowly clearing up, but the uppper lobe remained solidified. On the 21st, thirteen days after the crisis, the lower lobe had cleared up with practical completeness, but the upper lobe remained unchanged and was entirely solidified. Tubercle bacilli were absent from the sputum.

On this date the first x-ray exposure was given him, a picture being then taken which showed a shadow corresponding with the signs of solidification in the upper lobe. Two days later it was noted that the breathing was less harsh and the dulness less marked, but the signs were not strikingly changed. On the 26th the signs were decidedly less marked, dulness and bronchial breathing being much less pronounced. On the 28th there was still some dulness, but it was decreased. The breathing was blowing, but not distinctly bronchial. On March 1 the dulness reached only to the third rib, and was slight, the breathing still being somewhat blowing. On the 3d the signs were about as last noted. On the 6th there was still slight dulness to the third rib anteriorly. Posteriorly, the resonance above the spine of the scapula was skodaic. The breath sounds were somewhat harsh over the dull area, but not tubular. Rales were absent. The patient was discharged two days later, at his own request, in apparently normal condition, except for the signs mentioned. The x-ray exposures subsequent to the 21st occurred on the 24th, 26th, 27th, and 28th of February; and the 1st, 3d, 4th, 5th, 6th, and 7th of March.

Metabolism experiments were carried out in the two cases last described. As previous studies had shown no noteworthy influence of the x-ray exposures upon absorption from the alimentary tract the feces were not studied. The patients were given a diet composed of milk, bread, butter, eggs, rice, potatoes, and sugar. The food was from the same source each day and was accurately weighed or measured. The amount of nitrogenous intake was calculated from the weight records of the food, using figures that we had previously obtained from numerous determinations of the same food. The salt also was kept at the same amount each day, in order that variations should not influence the chloride determinations; and it should be noted that the diet was purin-free and hence that variations in the uric acid were due solely to changes in metabolism, and not to changes in the food. Before studies of metabolism were begun, the patients were put for three days on the diet described. For several days control studies were then made, and after this the *x*-ray exposures were commenced. Each "x" in the second column indicates an exposure; it is to be noted that the effect is shown in the figures of the following day.

TABLE I. CASE II.—URINE.

Date.	*x*-ray exposures.	Nitrogen ingested Grams.	Nitrogen.	Chlorides.	Uric acid.	Phosphates.
Jan. 12	...	6.54	8.971	12.600	0.2295	1.900
“ 13	...	6.54	5.262	8.262	0.1944	1.296
“ 14	...	13.08	14.840	11.125	0.3022	2.710
“ 15	...	13.08	9.908	13.360	0.3424	1.996
“ 16	x	13.08	8.153	12.350	0.2427	1.716
“ 17	x	13.08	17.875	20.900	0.3248	2.257
“ 18	x	13.08	Lost			
“ 19	x	13.08	10.819	14.630	0.3190	2.296
“ 20	x	13.08	11.464	14.490	0.3622	2.250
“ 21	x	13.08	9.222	14.170	0.2754	1.861
“ 22	x	13.08	8.119	16.250	0.3917	2.168
“ 23	x	13.08	9.863	17.250	0.3346	1.905
“ 24	...	13.08	11.752	16.880	0.3720	2.080
“ 25	...	13.08	7.207	14.560	0.2388	1.565

TABLE II. CASE III.—URINE.

Date.	*x*-ray exposures	Nitrogen ingested. Grams.	Nitrogen.	Chlorides.	Uric acid.	Phosphates.
Feb. 19	...	9.25	4.872	2.400	0.1260	0.720
“ 20	...	“	9.016	3.920	0.2835	1.169
“ 21	x	“	8.288	4.300	0.3000	1.170
“ 22	...	“	17.096	8.400	0.4662	2.380
“ 23	...	“	12.776	7.475	0.4215	1.794
“ 24	x	“	5.264	3.200	0.1380	0.795
“ 25	...	“	8.895	5.330	0.2007	1.404
“ 26	x	“	6.428	2.780	0.2120	0.888
“ 27	x	“	9.872	5.800	0.3757	1.430
“ 28	x	“	7.915	5.060	0.2268	1.408
Mar. 1	x	“	19.958	8.937	0.7137	2.614
“ 2	...	“	10.668	6.300	0.2962	1.800
“ 3	x	“	4.368	2.240	0.1440	0.552
“ 4	x	“	11.230	4.042	0.2756	1.764
“ 5	x	“	4.211	2.880	0.0740	0.920

Clinically then in these three cases the result was in each instance a rather rapid clearing up of most—in two cases practically all—of the signs of solidification that had previously persisted in the three cases, respectively, for one month, two weeks, and thirteen days, without improvement. The relation of the improvement to the *x*-ray treatment may have been merely a coincidence, for these exudates often, without any evident reason, clear up after more or less delay. Three cases do not suffice to decide this point, but that the *x*-rays played a lively part in causing the improvement appears to be at least probable, when one considers the influence that the *x*-ray exposures had upon metabolism.

In Case II, as a result of the first exposure (which was *only eight seconds long*, but was actually a fairly intense exposure, since it was one intended to produce a negative), the nitrogen increased to more than double what it had been before, and the chlorides showed an almost equally striking increase; the phosphates were somewhat less decidedly increased, while the uric acid showed relatively little change. The increase persisted in less-marked degree, gradually decreasing coincidently with the decrease of the solidification, and when the latter had disappeared, excretion had reached about its former level.

In Case III, the relation of the excretion to the *x*-ray exposures is even more striking. The first exposure was followed by a decided increase. Owing to a misunderstanding, the next exposure was not given for three days, and in the interval the excretion dropped decidedly; after the second exposure there was a moderate rise and then there was again an interval of two days between the exposures, and the second increase of excretion was also followed by a decided drop. The subsequent daily exposures were accompanied by an increase that was somewhat irregular, but became extremely remarkable on March 1. Again following the omission of an exposure on March 2, the urine of the 3d shows a striking drop, and finally, in this case also, as the solidification largely disappeared, the excretion appeared to be dropping pretty well to the original point, in spite of continued exposures, though, as the patient was soon to leave the hospital we could not carry the observations far enough to determine the latter point positively. At the end of the experiments in the two cases the first patient was retaining a great deal of nitrogen and the second patient was showing a strong tendency to retention; while in the periods during which they showed a marked increase in the excretion as the result of the *x*-ray exposures both were showing a very decided loss of nitrogen.

The figures that we have given show that the effect upon metabolism was indeed a most remarkable one. We have seen similarly profound effects only in one of the cases of leukemia that we have previously described—in which there was an extremely rapid improvement in general condition, splenic tumor, and leukocytosis—and in the two cases of general toxic reaction that we shall describe in another paper, though it should be noted that in the two latter cases the effect, while profound, differed strikingly from that in the case of successfully treated leukemia and in these cases of unresolved pneumonia.

Since such a remarkable effect was exerted upon metabolism, and since this was coincident with rapid improvement in the pulmonary signs, it would appear highly probable that the *x*-rays caused the improvement. The conditions observed are, in fact, extremely similar to those that occur at the time of an ordinary crisis in a favorably progressing pneumonia; that is, the exudate grows moist and clears up within a short time and, coincidently with this, there is a large increase in the metabolic output through the urine.

Whether the *x*-rays will prove to be of great importance in the treatment of unresolved pneumonia is, therefore, a question that is worth settling, but it is one for the future to decide. While, however, this decision is of importance, it seems to us that there is a broader interest in the question whether the theory that was the basis of these observations—the theory that the *x*-rays act upon metabolism chiefly as a ferment accelerator—is correct or not. When, for example, one administers hydrochloric acid with pepsin or alkalies with pancreatin, one consciously or unconsciously uses, to be sure, the same principle; that is, one attempts to provide conditions that are known to be especially favorable to the rapid action of these ferments; but in the manner in which these measures can now be used artificially, the results of such treatment do not at best distantly approach those accomplished in natural circumstances. Except in a few such simple and comparatively ineffectual instances, this principle of using measures that provide conditions that directly accelerate special ferment processes has not, to our knowledge, been deliberately used previously in the treatment of pathological states. In case our view is correct this study furnishes the first example of the deliberate use of this principle in the treatment of disease that lies beyond the digestive tract, and more especially the first instance of the conscious use of this principle with results that are so striking in their effect as to approximate those that the human organism itself accomplishes when successfully reacting to disease.

If, therefore, we are correct this is of considerable interest when one reflects upon the importance that ferment processes have assumed in the last few years in our conceptions of physiology and pathology. There is at present a widespread and somewhat excited tendency to give ferment processes a much more overwhelmingly important place than has been demonstrated to be their due; but the fact that they play an important part in the body economy is perfectly evident, and that methods that would clinically influence them in definite and controllable ways would be of great value scarcely needs to be stated.

Numerous attempts have been made to isolate and administer ferments themselves, of various kinds, in the treatment of diseases in which they are supposed to be absent; but such attempts have certainly been, in almost all instances, largely or entirely failures. This is scarcely to be wondered at, for in the first place knowledge of these ferments and of their relation to disease is in most instances at best very hazy, and, furthermore, attempts at isolating them are built upon most incomplete and insecure methods; and it is likely to remain most questionable whether tissue ferments can ever be obtained in active form, and then guided to the proper spot still in active form. It is equally questionable whether, should they arrive there, the disordered medium in which they would often find themselves would be such as to permit them to act. Indeed, it is exceedingly doubtful whether the whole idea of administering ferments is not as a rule wholly unphilosophical, both for the reasons given and because in many of those instances in which they appear to be absent they are probably not entirely absent, perhaps not even reduced in amount, but are merely unable to act properly owing to unfavorable chemical or physical conditions. It is possible that we may ultimately learn to stimulate the production of ferments in different organs with some accuracy, and this may prove to be occasionally a successful measure; but it is at present in the distance, and for the reason mentioned in the last sentence, it would frequently have only ineffectual results, for it is wholly rational to conceive of most varied chemical and physical conditions that would more or less completely interfere with the action of ferments in the tissues, and many such conditions, both simple and complex, are already known. If such conditions were present, increasing the amount of ferment would, of itself, be of no avail; and the essential matter would be to substitute for existing substances or conditions others that would inaugurate or accelerate the action of the ferment that is present. This is probably done already in using various therapeutic measures that have been evolved empirically; many drugs, for instance, are known to have a very marked effect upon a number of catalytic processes, and observations yet unpublished, that Dr. Casper Miller and Dr. Edsall have made, indicate that the striking lesions produced by toxic doses of mercury, and probably the remarkable effect that mercury exerts on syphilitic tissues, are, partly at least, the result of increased autolytic activities. The conditions under which a ferment acts are probably more frequently important in producing pathological states than is the amount of ferment present, for ferments are exceedingly sensitive to the conditions under which they act, in both a positive and a negative way, and the conditions influencing them are exceedingly numerous and varied. The almost inconceivably energetic effect of conditions that have a very marked accelerator action is shown, for example, by the fact that the intestinal wall appears to be able in the brief period that is necessary for their passage through it, to rebuild into the native protein of the individual the fragments of protein broken down in digestion, while the best conditions that experimenters can now provide furnish, even after long periods of action, at most, only doubtful evidence of slight artificial imitation of such a process; and, on the other hand, as is well known, mere traces of certain substances, such as hydrocyanic acid, will greatly retard or wholly interfere with the action of relatively enormous quantities of some catalyzers.

This casual mention of some of the most evident conditions that are known to influence the action of ferments suffices to make it reasonably clear that if a study of ferments is to become of value clinically it is likely to be chiefly through determining the conditions that influence the activity of individual ferments under normal and pathological conditions and through making use of measures, clinically applicable, that can be shown to provide, of themselves and directly, favorable conditions for the action of these ferments, or that overcome unfavorable conditions.

Whether our view regarding the action of the x-rays and their effect in unresolved pneumonia is correct or not the future will probably see the principle upon which we were working used many times, as knowledge of ferments and of their accelerator and anti-accelerator conditions increases. That which has just been said must not, however, be misinterpreted. It is intended largely as a protest against the blind but common belief in a mysterious value of ferments as therapeutic agents, a faith that gives opportunity to manufacturers to foist upon many of the profession preparations that are even supposed to contain, isolated and in active form, ferments that are by no means definitely known to exist; and still more would we protest against the excited enthusiasm of those investigators whose work has been such as to lend them the respectful attention of the profession, and who now seem occasionally to be heralding the approach of a therapeutic millenium that is to be distinguished chiefly by the administration of ferments. We would not be thought to believe, however, that there is, instead of this, a nearer-by millenium, in which knowledge of the conditions in which ferments act will lead to inevitable success in the treatment of disease. We would merely point out that in contrast with the dubious plan of treating disease by administering ferments, those who think conservatively in regard to this matter can scarcely believe that much will ever be accomplished in a large proportion of cases in which ferment action is at fault except by modifying the conditions under which ferments act. Through happy chance it will not improbably occur that occasionally the knowledge that accumulates will be made of service in successful therapeutic practises, if this manner of viewing the relation between ferments and therapeutics is held in mind; but any such success will almost certainly, for a long time to come, be largely the result of happy chances that are pointed out by a modicum of suggestive investigation. It will surely be a wearily long period before slow, painstaking, and complex investigations make it possible to apply any such principles in a considerable number of cases with a just expectation of success. The exciting work on ferments that has occurred in recent years has greatly transformed the point of view assumed in attacking problems in pathology, but has not in other ways profoundly simplified these problems. The difficulties and complexities have not been essentially lessened; their nature has rather been made more evident.

A BRIEF STUDY OF A DIPHTHERIA EPIDEMIC AT THE ADIRONDACK COTTAGE SANITARIUM (FOR INCIPIENT PULMONARY TUBERCULOSIS).

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and

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Diphtheria epidemics in institutions for the tuberculous are of unusual occurrence, for which reason, as well as on account of the many problems that arise under such conditions, it has been deemed of sufficient interest to report in more or less detail a recent epidemic of diphtheria at the Adirondack Cottage Sanitarium.

The origin of the first case has never been clearly determined. We present the facts as they occurred. Early in October, 1905, one of our orderlies, after a visit to his home, developed a membranous tonsillitis, which on a single culture showed streptococci and no diphtheria bacilli. He was allowed accordingly, after a few days' isolation, to resume his duties. Several cultures taken from his throat at intervals later on during the epidemic, as well as those from his room-mate, were always negative. In the latter part of the month, one of the maids, who had but recently begun work in the laundry and who was with the orderly quite frequently, reported with a well-developed tonsillar diphtheria. She was promptly isolated in her room with her room-mate, her sister, and a strict—as we thought—quarantine established. Later we learned that, standing in her doorway, she would hold conversation with the other maids. After most rigid precautions, including negative cultures, both sisters were discharged, the latter on Saturday evening December 30, 1905. The same day, before the first maid was discharged from quarantine, a patient and one of the maids had a slight soreness of the throat, and two days later when they reported that they felt ill, a second patient went to bed. The second maid, who was employed in the dining-room and linen-room, was at work until she went to bed in the evening, when the diagnosis was made in all three. The patients were isolated and given antitoxin at once. The next day a cottage entirely apart from the other buildings was opened as an isolation pavilion, placed in charge of a competent nurse, and the patients transferred there. The same day, January 2, the throats of all in the institution were carefully inspected, and a patient and two maids with tonsillar membranes were isolated. One of these was probably not diphtheria, although a positive culture was obtained at a later date. The next day, January 3, all patients and employees, irrespective of any pulmonary or other trouble, were given an immunizing dose of 500 units of the antitoxin prepared by the New York State Board of Health. The two cottages and the Childs Memorial, the infirmary where the cases of diphtheria had occurred, were isolated. Each inmate of these cottages was given 1000 units, and not allowed to leave the cottage for one week. During this week several (six) cases of follicular tonsillitis developed, which, we might add, caused much uneasiness, especially as one occurred in one of the infected cottages. The infirmary patients received but 500 units, and one of them who had complained of sore throat developed a membrane on the posterior pillar. She was promptly transferred to the isolation cottage, but the membrane proving by culture to contain only streptococci, she was sent back to the Childs Memorial. It was with some trepidation that immunizing doses were given to five of the infirmary patients, as they were in an advanced stage of tuberculosis, and in three cases the disease was more or less acute. No more cases developed until January 16, two weeks after the first immunizing dose was given, when one of the nurses developed a rather sharp attack of tonsillar diphtheria. The day following, January 17, the fireman, and on January 18 and 19 two patients were attacked. On January 20 a second immunizing dose of 500 units was given to all patients who had not shown especial susceptibility to the antitoxin. To these a dose of 400 units of purified antitoxin prepared by the New York City Board of Health was given a few days later. No more cases of clinical diphtheria developed, but on January 21 it was deemed expedient to take cultures from the throat and nose of every inmate of the institution. The result was appalling, twenty-two of the patients and nine of the employees, including the chef and the baker, revealed positive cultures. The attempt at isolation of inmates with positive cultures, but without clinical symptoms, which had been begun, was gloomily abandoned. The health authorities quarantined the institution, but strange to say few patients became frightened. On January 24 the last case, a patient whose throat was negative at first, developed an apparent follicular tonsillitis which was positive on cultures, and was isolated. A second cottage had been opened and a nurse placed in charge. A third and last immunizing dose (400 units of the purified antitoxin) was given on February 3. The quarantine was raised February 20, after the last case of clinical diphtheria showed two successive negative cultures. No new patients were admitted until February 27, and for some weeks each was given 400 units of antitoxin on admission.

To summarize briefly, the epidemic lasted fifty-one days, that is, from January 2, when three patients became ill, to February 20, when the last patient was discharged from quarantine. Eleven cases of clinical diphtheria developed, and in 32 other individuals diphtheria bacilli were found on culture. There were no fatalities and no serious complications. In all over 400 immunizing doses were given to 174 individuals—129 tuberculous and 45 nontuberculous.

Epidemics of diphtheria in institutions are by no means rare, but we can find no record of such an epidemic in a sanitarium for pulmonary tuberculosis in which the youngest patient was aged about eighteen years. Such a condition presents many peculiar phases and problems on which we could get little or no information. In the first place, should the patients be given immunizing doses of antitoxin? How often should these doses be repeated? Should cultures be taken from the throat and nose of all the patients or only from those with clinical diphtheria? How should the treatment for the pulmonary tuberculosis be modified, if at all? Should the pulmonary tuberculosis modify the amount of antitoxin in the diphtheria?

The most important point in prophylaxis is complete isolation. We feel now that had we set aside a separate cottage and placed in it the first patient under the supervision of a competent nurse the epidemic would have been avoided. We took every precaution, but it is difficult to impress the necessity of complete isolation upon many persons, not only those of low intelligence. For this reason a nurse is a *sine qua non*. The most careful quarantine was established as soon as the epidemic broke out. No measures were omitted. Every room was carefully fumigated with formaldehyde and completely renovated, and each of the cottages where diphtheria occurred was scrubbed down with corrosive sublimate, revarnished, and retinted.

Immunizing Injections. All inmates received two or three immunizing doses of antitoxin. The doses were at first 500 and later 400 units.

A small amount of antitoxin prepared by Parke, Davis & Co. was used, but the greater part was from the New York State and City Health Departments.

The injections were made for the most part midway between the scapula and the spines of the eighth and ninth dorsal vertebræ. Many of the women preferred the injection in the arm, but the local reactions were much more severe in that region.

Preparation. The preparation of the skin can be done very simply. It was cleansed with absorbent cotton, usually first with soap and water, then always, and in some cases only, with alcohol. The site of injection was closed with collodion. The most convenient syringe we found to be the small "Sub—Q" glass syringe, with an asbestos wrapped glass plunger, holding about 2 c.c. Small needles were used, and the whole syringe was frequently boiled; after each injection they were rinsed out with boiling water.

Results. In no case did any infection occur. In four instances diphtheria developed between the fourteenth and sixteenth day following the first injection, and in one case four days and in another one day after the second injection. It was therefore deemed advisable, when only 500 units were used, to repeat the injection for two or three times at intervals of two weeks.

The purified antitoxin produced much fewer unpleasant symptoms than the other varieties, which in many instances produced general malaise, more or less rash, swelling of the face, hands, and feet, pain in the joints (so severe in two cases as to cause insomnia and to require morphine), stiffness of the joints, nausea and vomiting, as well as in nearly every case more or less local tenderness and slight swelling for a few hours.

The injections were usually made in the forenoon and the malaise, when present, usually occurred within twenty-four hours. The majority of the patients suffered some slight inconvenience, but in most cases it was of no moment. In some patients it was postponed for eight to fourteen days and occurred simultaneously with the rash.

The rash occurred more in women than in men, and less in the tuberculous than in the non-tuberculous. Of 140 cases, 32 (23 per cent.) had some rash. In 16 cases the rash was severe. The usual form of rash was urticaria, but fine pin-point papules were present in some cases, more often at the site of the injection. A few acne-like papules occurred in a few cases, chiefly on the face and in women. In two patients who had previously suffered from purpura, a crop of purpuric blotches appeared after the injections. No rash occurred after the first or second injections in 92 patients. In 9 it occurred only on the first, in 6 only on the second, and in 4 only after the third immunizing dose. From this it would appear that the rash is as likely to occur after the first as after the second or third injections. Of 10 non-tuberculous individuals the rash appeared on the seventh day in 5, 1 each on the first, second, eighth, and thirteenth days. Among 21 tuberculous cases, the rash appeared as follows: second day, 2; fourth 3; fifth, 1; seventh, 3; eighth, 5; tenth, 5; fourteenth and seventeenth, 1 each. The rash, therefore, occurred more frequently between the seventh and tenth days, but it may occur as early as the first and as late as the seventeenth day after injection.

The pulse was quickened in several patients. One suffered from palpitation, in another an intermitting pulse was present. The temperature was raised in thirteen patients and possibly more would have shown this symptom had careful records been kept of each patient. In no instance was the temperature raised for more than a few days. In three patients who were in bed on account of elevated temperature the injections produced no noticeable effect on the temperature further than to raise it a fraction of a degree for a few hours. Stiffness in the joints of the hands, knees, and in several instances in the hips, was present. Associated often with the stiffness and possibly its cause in some instances was swelling of the face, hands, and feet. In many cases the swelling was more or less localized in the arm or leg where the injection was made. Marked swelling occurred in thirteen cases.

The axillary glands were enlarged in five cases in which the injections were made in the back. In one case a chain of enlarged and tender glands extended to the breast. Diarrhœa occurred in one case, chills in another, and pleurisy in a third. In two cases, hemoptysis occurred about one week after an immunizing dose. In some of the foregoing instances the "complications" were no doubt mere coincidences and we believe this to be true more especially of the hemoptyses.

THERAPEUTIC INJECTIONS. The tuberculous were treated when they developed diphtheria, exactly as the non-tuberculous; but when possible they were placed in bed out-of-doors and they slept and lived in rooms with windows wide open at all times. The antitoxin was given in doses of 2000 units daily in the thighs until the symptoms and membrane were markedly affected. In three patients no rash developed, but one, a tuberculous patient, had besides the two immunizing doses of 500 units only one large dose, 4000 units. The rash in the other eight was urticarial, severe and general in six, confined to the legs in two.

One patient, a non-tuberculous case, had some cardiac weakness, but no other complications occurred. The patients, with but two exceptions, received large doses on the first day of the appearance of symptoms, when the membrane was wholly confined to the tonsils. The membrane disappeared in two non-tuberculous cases in five and seven days, in five tuberculous cases, in three, three, five, six, and seven days respectively.

No immediate effect upon the tuberculous process was noted in any cases. The fever disappeared as quickly as in the non-tuberculous. The cough was not increased nor did any patient have night sweats or hemoptyses, though an occasional pleuritic pain was complained of, which, however, was not out of the ordinary. A careful examination of the lungs a short while after discharge from quarantine revealed in all cases an improvement, more or less marked, in the pulmonary condition.

CONCLUSIONS. From our experience in this mild diphtheria epidemic we would conclude that:

1. In institutions immunizing doses of antitoxin of 500 units should be given every two weeks for two or three times.
2. Diphtheria may occur in patients two weeks after an immunizing dose of 500 units.
3. It is unnecessary to endeavor to separate individuals with positive cultures from the nose or throat without clinical symptoms from those with negative cultures when immunization is carried out.
4. One negative culture from the throat is sufficient neither for diagnosis nor for raising quarantine.
5. The advisability of taking cultures from the nose except in nasal diphtheria may be questioned.
6. Careful isolation for three or four weeks under a trustworthy nurse should be practised. After that time the patient with a positive culture should be kept out of the general dining-room or buildings and at a distance of four feet from all non-diphtheritic patients.
7. Some untoward symptoms, usually slight, appear in most patients. A rash, arthralgia, swelling, and general malaise occur most frequently. Slight transitory rise of temperature occurs frequently.
8. In institutions where the out-door treatment is rigidly enforced the epidemic will probably be cut short.
9. In the presence of a diphtheria epidemic, patients with pulmonary tuberculosis should be treated as regards the diphtheria as otherwise healthy individuals.
10. When the tuberculous contract diphtheria they should be moved out-of-doors as soon as possible.
11. Mild diphtheria seems to exert little or no harmful influence upon the pulmonary lesion in the tuberculous.
12. The antitoxin in small or large doses seems to have little or no effect upon the pulmonary tuberculosis.
13. Immunizing doses may be given to patients with fever.
14. Complications seem to occur after diphtheria no more often, possibly less, in the tuberculous than in the non-tuberculous.

We desire to record here our hearty appreciation of the kindness of the officers of the New York State and City Boards of Health. We feel that it is but just to say that without their assistance and advice and the promptness with which they supplied us with antitoxin this mild epidemic might have been most serious. Our acknowledgments are due also to the health authorities of the village of Saranac Lake and of the township of St. Armand.

THE CHEMICAL AFFINITY OF MUCUS FOR HYDROCHLORIC ACID.[1]

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Some months ago, with the co-operation of Dr. A. V. S. Lambert, I began a study of the secretion of HCl under various conditions. Dogs were used, and the gastric juice was obtained by means of Pawlow fistulæ. In the course of the experiments it was noted that while the gastric juice collected from the artificial stomach had no opportunity to come in contact with the food, yet a considerable fraction of the HCl always existed as combined acid, whereas all the acid apparently should have been uncombined free acid. At first this result was supposed to be due to some serum which had run into the collecting funnel from the stitch margin of the fistula along with the gastric juice; but a month later, when the wound was healed, this explanation did not appear adequate. It had also been noted that the mucus in the collecting receptacle varied from hour to hour, and that when the amount of mucus was considerable the combined acid was also increased.

In his studies of the secretion of acid in the stomach Pawlow observed a fluctuation which he thought might be explained by different rates of flow—that is, when the gastric juice was rapidly secreted it passed quickly over the mucous membrane and was not so much "neutralized" as when the secretion was slow. Pawlow speaks of the mucous membrane as "alkaline." I am unable to determine from his papers whether Pawlow determined the total amount of HCl in the gastric juice as well as the free acid, but I am led to think he did not, because of his conclusion that the amount of HCl secreted in the stomach during the digestive period is constant. I am unable to find any reference in the papers of Pawlow or his pupils relative to mucus as a "neutralizing" medium for HCl in the stomach.

The function and fate of the mucous secretions in the various parts of the gastrointestinal canal are relatively unknown, in a physiological as well as a chemical sense. In the stomach mucus is constantly being secreted to some extent, but more at some times than at others, as, for example, at the end of a digestive period; and in certain pathological conditions the amount of this secretion is quite considerable.

Several years ago it was observed in this laboratory that "precipitated mucoid shows practically no combining power with acids. In the hydration of mucoid by pepsin acid, however, acid combines

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[1] Received for publication September 30, 1906.

with the dissolved protein products formed in the process."[2] It seemed probable that similar behavior of the gastric mucus accounted for the constant occurrence of combined acid in the juice obtained, and at Gies' suggestion the following experiments were conducted along lines similar to those that had been performed on mucoid.

Mucus from pigs' stomachs was used.[3] In the first series of determinations this mucus was dissolved in 0.5 per cent. Na_2CO_3. The solution was filtered, neutralized with dilute HCl, and precipitated by means of alcohol. This precipitate was collected on a filter and washed with water. Mucous protein obtained in this way is only slightly soluble in water and is neutral in reaction.[4] A suspension in water was made of this mucous product, and to it was added ^{3n} HCl until the solution gave just a faint reaction for free acid with Günsberg's reagent. Under these conditions, while there is some combining affinity of mucous protein for HCl, it is insignificant.

On the other hand, when 0.1 gram of pepsin[5] is added to such a mucous suspension the result is marked, as shown in the following summary of data obtained in one of the experiments:

Table I.

Aqueous suspension of about 1 gram of dry mucous protein and 0.1 gram pepsin. Titration with $\frac{3}{20}$ normal HCl.

1.4 c.c. produced reaction for combined acid.

1.6 c.c. produced faint reaction with Günsberg's reagent.

1. After digesting 24 hours in a thermostat at 40° C. there was no free acid present. 2.7 c.c. $_{3n}$ HCl added.
2. After digesting 48 hours.[6] No free acid present. 0.5 c.c. $\frac{3n}{20}$ HCl added.
3. After digesting 72 hours. Free acid present.
4. After digesting 96 hours. No free acid present. 2.5 c.c. $\frac{3n}{20}$ HCl added.
5. After digesting 120 hours. Free acid present.
6. After digesting 144 hours. No free acid present. 2 c.c. $\frac{3n}{20}$ HCl added.
7. After digesting 168 hours. No free acid present. 2 c.c. $\frac{3n}{20}$ HCl added.
8. After digesting 192 hours. Free acid present and persistent.

Total acid added 11.3 c.c.

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[2] Gies, Proceedings of the American Physiological Society, p. xiii; Amer. Jour. of Physiol., viii, 1903. Also Gies and collaborators, Biochemical Researches, 1903, i, p. 54.

[3] It was obtained mechanically by lightly scraping the surface of the mucous membrane.

[4] The relation of this result to the studies of salts of mucoids, nucleoproteins, etc., now in progress in this laboratory, will be pointed out later.

[5] Parke, Davis & Co., 1 to 2000.

[6] All records of time in these summaries designate *total* periods from the beginning of digestion.

Table II.

Control conditions same as in Table I. 2 c.c. $\frac{3n}{20}$ HCl added.
1. After digesting 24 hours. No free HCl. 2 c.c. $\frac{3n}{20}$ HCl added.
2. After digesting 48 hours. No free HCl. 2 c.c. $\frac{3n}{20}$ HCl added.
3. After digesting 72 hours. No free HCl. 4 c.c. $\frac{3n}{20}$ HCl added.
4. After digesting 96 hours. Free acid present.

Total acid added 10 c.c.

The mucus-hydrochloric acid mixtures in these two experiments were combined, and, when filtered and after being neutralized, were submitted to tests for the products of digestion. These digestive products consist almost wholly of proteoses, with only a trace of peptones.

A second series of observations was made in which, instead of the precipitated mucous protein used in the previous experiments, I employed the fresh mucus as it is immediately after its removal from the pig's stomach. The observations were not intended to be strictly quantitative, but they served well the chief purpose in view, that is, to ascertain whether fresh mucus combines with acid. The mixtures were kept well covered with toluol and the digestion carried on at a temperature of 40° C. The results were as follows:

Flask I.

Mucus, 2 grams; water, 25 c.c.
Pepsin, 50 mg.; ^{3n} HCl, 4 c.c.
1. 24 hours. Free acid present.
2. 48 hours. No free acid present. 2 c.c. $\frac{3n}{20}$ HCl added.
3. 72 hours. Doubtful reaction. 1 c.c. $\frac{3n}{20}$ HCl added.
4. 96 hours. Free acid present.
5. 120 hours. Free acid present.
6. 144 hours. Free acid present.

Total acid added 7 c c.

Flask II.

Mucus, 2 grams; water, 25 c.c.
Pepsin 100 mg.; $\frac{3n}{20}$ HCl, 4 c.c.
1. 24 hours. No free acid. 2 c.c. $\frac{3n}{20}$ HCl added.
2. 48 hours. No free acid. 2 c.c. $\frac{3n}{20}$ HCl added.
3. 72 hours. Free acid present.
4. 96 hours. No free acid present. 2 c.c. $\frac{3n}{20}$ HCl added.
5. 120 hours. Free acid present.
6. 144 hours. Free acid present.

Total acid added 10 c.c.

Flask III.

Mucus, 2 grams; water, 25 c.c.
Pepsin, 200 mg.; $\frac{3n}{20}$ HCl, 2 c.c.
1. 24 hours. No free acid 4 c.c. $\frac{3n}{20}$ HCl added.
2. 48 hours. Free acid present.
3. 72 hours. Free acid present.
4. 96 hours. No free acid. 2 c.c. $\frac{3n}{20}$ HCl added.
5. 120 hours. Free acid present.
6. 144 hours. Free acid present.

Total acid added 8 c.c.

Flask IV.

Pepsin, 100 mg.; water, 25 c.c.; $\frac{3n}{20}$ HCl, 4 c.c. No mucus.
1. 24 hours. Strong reaction for free acid.
2. 48 hours. Strong reaction for free acid.
3. 72 hours. Strong reaction for free acid.
4. 96 hours. Strong reaction for free acid.
5. 120 hours. Strong reaction for free acid.
6. 144 hours. Strong reaction for free acid.

The pepsin-acid solution was titrated against $\frac{3n}{20}$ NaOH in the last case, 3 c.c. being required to neutralize the free HCl present. The 1 c.c. which had been found was probably in combination with some proteid material such as proteose that existed as impurity in the pepsin preparation.

It is clear, I think, from these results, that mucus, like albumin and other proteins, is capable, in the presence of pepsin, of combining with hydrochloric acid. This is true not in a slight degree, but to a relatively marked extent. It is probable, also, it seems to me, that in the stomach the combining power of fresh mucus, as it is secreted from the gland, is much greater than these experiments indicate, because in using, as I have had to do, mucus removed from the stomachs of pigs that had probably not been recently fed, a hunger stimulus would have excited the flow of some gastric juice, and thus would largely have satisfied the combining power of the mucus used in the experiments.

However that may be, the combining power of mucus with HCl, as demonstrated in these experiments, is sufficient to explain some of the physiological and pathological phenomena that are well recognized. For example, in certain morbid gastric conditions there is observed a marked diminution in the amount of free HCl, while the total acidity (exclusive of organic acids) is often almost or quite normal. Along with this there is an excessive secretion of mucus. In such states it does not seem unlikely that the combined acid is bound with mucus, and the condition resembles that observed in dogs with Pawlow fistulæ, in that during the later periods of digestion mucus is secreted abundantly, and often at such times there is only a trace of free HCl, while the total acidity may be considerable.

REVIEWS.

American Practice of Surgery: A Complete System of the Science and Art of Surgery. By Representative Surgeons of the United States and Canada. Edited by Joseph H. Bryant, M.D., and Albert H. Buck, M.D., of New York City. Complete in eight volumes. Vol. I, pp. 818. New York: William Wood & Co., 1906.

Surgery, its Principles and Practice. By Various Authors. Edited by William Williams Keen, M.D., LL.D. In five volumes. Vol. I, pp. 983. Philadelphia and London: W. B. Saunders Co., 1906.

The appearance of the initial volumes of two *Systems of Surgery* within a few months of each other naturally raises a query as to the utility in general of such treatises. The mere fact that two systems are being published simultaneously does not prove that there is need for both, yet it is at least evidence that there are good prospects of each meeting with a cordial reception, since works of such magnitude are seldom undertaken without assured support.

There are two chief forms in which surgical literature of a permanent kind is placed before the public—the monograph and the system. While the author who presents his views in the form of a monograph rarely is recognized as such an authority during his lifetime as is the author or editor of a system, yet, in years to come, his work advances to a higher niche in the temple of fame, while the system is replaced, discarded, even forgotten. Who reads Heister or Boyer? Who does not read Malgaigne, Paget, Gurlt? The reason for this difference, we venture to think, is that the chife value of a "system" consists in its being systematic—that is, in the fact that all subjects of surgery are included in its volumes, the little as well as the great, the rare diseases as well as those of frequent occurrence. In a monograph the author deals with one subject or at most with a group of subjects closely related; and his personality lends character to the whole. In a system the text should be cyclopedic in character. Hence it is that skilled taxonomists make the best editors; and on this account mainly, we think, do the volumes at present under discussion fall short of the standard which the reviewer has in mind.

The first of these two volumes claims for itself a distinctively

American character; and, while we have no sympathy with that narrow provincialism which prefers an American to a foreign work merely because it is American, yet we feel no fair-minded critic can deny that representative American surgeons are at least as well qualified to write upon surgical topics as are those of any other single nation. This, however, is not asserting, and indeed we do not admit, that a still better system could not be produced by the judicious selection of authors from European as well as American countries.

We do not believe that an editor's labors consist solely in outlining the plan of a work and in selecting authors for the various articles, and then turning the authors loose into the field of surgery to wander where and at what length they will. We hold that the editor should personally supervise the construction of each article, and so correlate one to the other that a finished whole shall ultimately be presented to the reader. Keen, who in this arduous task has had the efficient aid of J. Chalmers DaCosta, fulfils, in these respects, the duties which we think appertain to an editor more successfully than do Bryant and Buck.

In the *American Practice of Surgery* the basic principles are discussed in five parts: I. Inflammation (Warthin); disturbances of nutrition (Nicholls, of Montreal); repair (E. H. Nichols, of Boston); tumors (Nicholls); theories of tumor formation (McGraw); parasitical relations of cancer (Gaylord). II. Infections of occasional occurrence (Paul M. Pilcher, of New York)—a thoroughly unscientific contribution; surgical shock (Bloodgood)—much superior to Crile's parallel contribution in Keen's *Surgery*. III. Surgical diagnosis (Bryant)—a diffuse and rambling essay; body fluids in surgery (Harlow Brooks); radiographic interpretation of epiphyses (Hickey); technique of radiography (Osgood, of Boston)—a very technical paper. IV. General surgical treatment (J. E. Moore). V. Prognosis (Leonard W. Bacon, of New Haven).

The first volume of Keen's *System* includes: Physiology (Crile); hematology (Da Costa)—a needlessly elaborate and technical paper; infection and immunity (Hektoen)—orderly, concise, intelligible; inflammation (Adami); suppuration, abscess, ulcers, gangrene, etc., (Freeman); repair (Francis Carter Wood); thrombosis and embolism, erysipelas, tetanus, scurvy, special infections, and poisoned wounds—articles by Frazier, much in the manner of his recent contributions to *Progressive Medicine;* traumatic fevers (Eugene A. Smith); rickets (Nichols, of Boston); surgical tuberculosis (J. Chalmers Da Costa); chancroids and syphilis (Edward Martin); tumors (Bland-Sutton); wounds and contusions (Crile).

Each volume opens with an historical sketch, that in the *American Practice of Surgery*, by Stephen Smith, being limited to the evolution of American surgery, while Mumford, of Boston, in the other work begins his tale from the twin egg of Leda. Smith's article is sufficiently interesting, but possesses a biographical style which is wearying to the reader, and there are some inconsequential blunders which are an annoyance to anyone who loves accuracy of statement. One such is his constant reference to the University of Pennsylvania by the name of "University Medical College, Philadelphia." Mumford's style is distinctly individual, and at times decidedly undignified. His hortatory expressions are such as will give cause for offence to many students, while in others they will produce a derisive curl of the lip. His facts are not always exact, and we would caution the inexperienced reader against accepting as gospel truth the statement that Rhea Barton is remembered chiefly for his osteotomy of the *knee*. The reference on the same page to McClellan's operation for removal of the entire scapula and clavicle is inaccurate, since this operation was an "amputation above the shoulder." His citations of the titles of medieval Latin works show a curious fondness for inversions in the order of words. To devote fourteen pages to John Hunter and his times, with twenty-two pages to all preceding and twenty-four to subsequent times, seems ill-balanced. Surely Maisonneuve is more worthy of mention than von Kern and von Walther; Bonetus is not mentioned at all; Morgagni is merely named; and though a portrait of Astley Cooper is given, his career is disposed of in two lines, and that of le grand Petit in one!

Inflammation is discussed by Warthin in the *American Practice of Surgery,* and by Adami in the system edited by Keen. While Warthin's article is sufficient, we cannot but feel that its distinguished author is capable of better work. The subject is treated in too hazy and uncertain a manner for us to consider the article practical; yet there is too little profundity of thought for it to be classed as a masterpiece in the realm of theory. It has impressed us as the work of a man who, although perhaps an excellent teacher, yet lacks the pen of a ready writer—one who cannot lucidly express either his own thoughts or those of other people. His mind appears to be convinced of nothing; instead of giving its due weight to each of the factors usually recognized as playing a part in this complex process, he gives them all nearly equal weight, so that his article is like the concluding chapter of Rasselas, in which nothing is concluded.

Few things really new have been added to the pathology of inflammation since the day of Metchnikoff's discovery. Probably the most important is the bactericidal action of the blood plasma and of the fluid in exudates; and the next, the discovery of rudimentary lymph nodes throughout the tissues which supply a large proportion of the cellular elements found in a part which is inflamed. This source of supply is more emphasized by Warthin than by Adami, who, however, accepts as proved that much of the round-cell infiltration has its origin in proliferation of endothelial cells of one kind or another. Both these authors classify tuberculosis, syphilis, etc., as inflammations with special characteristics due to the cause and to the tissues affected. Though this is an unusual view to take at the present day, it is one which has always appealed to us as logically correct. It is, however, a view which is categorically denied in other portions of both volumes. The writers on inflammation also consider granulating wounds identical with ulcers, while a very emphatic denial of this dictum is found later in the volumes.

Such contradictions, moreover, are not confined to purely theoretical considerations. Thus we find Smith, of Buffalo, recommending, in Keen's *Surgery*, a separate building for the hospital treatment of erysipelas, while Frazier, in the same work, expresses the opinion that it is safe to treat it in the general ward with other infected wounds. For our own part, we think the former opinion in each case not only saner, but more safe, and join with the philosopher when he exclaimed: "*Errare malo cum Platone, quam cum istis vera sentire.*"

In Bryant and Buck's work the article on tumors is by Nicholls, of Montreal; in Keen's volume it is by Bland-Sutton. It is said to be a maxim of the civil law that definitions are hazardous, and in no respect, perhaps, is this more evident than in attempting to define a tumor. Nicholls agrees with Thoma in calling a tumor an independent new-growth. Bland-Sutton is too wary to be trapped into definitions of any kind. His article, one of the best in the volume, deals with the question in a broad way, and does not descend to many details. He makes little attempt at classification, but discusses in his usual masterly and comprehensive manner the clinical course and the symptoms of the various well-recognized types of tumor. His aim is to reduce the number of new-growths, and he is apparently of the opinion that tumors differ from the infectious granulomas only in the same way as these latter differ from ordinary inflammation. Indeed, to anyone who reads Stricker's studies in the pathology of inflammation, with the hindsight derived from a knowledge of Coley's work in the serum-therapy of tumors, it is not very difficult to detect the inkling of such an idea in the writings of the great Austrian pathologist himself.

Nicholls' article on tumors is much concerned with the subject of classification, which is the most important question of all at present, because only when we have them classified can we have any accurate idea as to their cause. He gives Adami's classification of tumors as an improvement upon the embryological classification of Waldeyer, but does not adopt it. Although this comparatively new classification appeals to us as philosophically correct, we quite agree with Nicholls that it is too complex to be adopted for general use at present. We cannot, however, agree with Nicholls that the classification which he does adopt presents any advantages whatever, except that of allowing tumors to be described very nearly in any sequence that may happen to strike the fancy of the writer.

McGraw supplements the article by Nicholls with a concise but thorough paper on theories of tumor formation. The sum of our knowledge is well expressed in his phrase that Nature abhors a cellular anarchy. Gaylord still further elaborates this subject by a temperate and conservative article on the parasitical relations of cancer. This whole paper, like most in the volume, might well be condensed into less than half its space, yet the argument is complete as it is, and perhaps would have less force if more succinctly stated. It is encouraging to note that as Gaylord's experience increases he is less ready to believe every unusual feature he sees in the blood to be a parasite of cancer. The readers of this JOURNAL will compare with surprise his present views with those which he expressed in its pages not many years since.

Yet, with all his conservative statements, Gaylord comes very near, we think, to proving the parasitical origin of cancer, save for the "if" with which his statements begin: "If a primary cancer," he says, "starts from a given centre and the cancerous transformation spreads from cell to cell, it must be that that force or factor which endows normal epithelium with the power of limitless proliferation is transferred, at least for a certain period of time, from the involved cells to the adjoining normal ones." Although he quotes Orth as supporting similar views, it seems to us more rational to suppose that cancer is propagated by proliferation of already diseased cells, rather than by infection or metaplasia of healthy cells; and as the interpretation of microscopic changes is confessedly difficult, we must still look upon this conclusion as dependent upon a very large If.

We have left ourselves, much to our regret, no space to speak of the excellencies of these volumes. If our review seem to some unduly critical, we can only plead in justification our view that the good points speak for themselves and that a reviewer's function is rather to discover shortcomings than to cover all with a gloss of indiscriminate praise. We would like to speak of Da Costa's article—the only truly encyclopedic contribution in either volume—on surgical tuberculosis, in Keen's *Surgery;* and of the novel and effective paper in Bryant and Buck's system on the radiographic interpretation of the epiphyses, by Preston M. Hickey, of Detroit; as well as of other articles too numerous to mention. There is a mine of information within these covers, not always in easily accessible form, nor is it always the purest ore; but such as will well repay earnest and conscientious study. A. P. C. A.

THE EYE AND THE NERVOUS SYSTEM. THEIR DIAGNOSTIC RELATIONS. By VARIOUS AUTHORS. Edited by WILLIAM CAMPBELL POSEY, M.D., Professor of Ophthalmology in the Philadelphia Polyclinic and College for Graduates in Medicine; and WILLIAM G. SPILLER, M.D., Professor of Neuropathology and Associate Professor of Neurology in the University of Pennsylvania. Illustrated. Pp. 988. Philadelphia: J. B. Lippincott Co., 1906.

IN estimating the value of a large and important book like this, the reviewer naturally strives to judge of its merits from several points of view. Does it really fill a gap? Does it successfully carry out the plan laid down in the preface? Does it make contributions of value to our knowledge? Is it uniform in its excellence and free from marked defects? Does it compare favorably with other books of its own class? Some of these questions require a careful answer, but it may fairly be said that, on the whole, the work will be welcomed as a clear, practical and conscientious discussion of an important group of subjects. As the sense of vision played such a large part in the evolution of our minds and brains, so the diseases which centre on the visual apparatus are of immense importance, both in their practical and in their scientific aspects, and there is no other treatise in our tongue that presents the various matters here at stake in so comprehensive and so modern a form as this.

The book with which one naturally compares it is the monumental work *Die Neurologie des Auges*, by Wilbrand and Sänger. It must be admitted that, so far as they go, the three parts which comprise all that has thus far been published of this last-named work are so searchingly thorough, and gain so much by being practically written by the same hand, that they place it among monographs of a higher order than most of those which make up the volume now before us. On the other hand, the German work is far more limited in its scope, and, thus far at least, practical questions of treatment are not discussed in it at all.

As has been said, Posey and Spiller's volume considers the diseases involving the eye and vision from a variety of standpoints, and it may be added at once that while this is one of the merits of the book, it is also one of its defects. Several long chapters might easily have been curtailed without any real sacrifice of their value for the main points at issue. This is true, for example, of the articles on tumors of the brain and on the diseases of the pons, although these articles, which are complied with admirable care by Dr. Spiller and Dr. E. W. Taylor, are exceedingly interesting and valuable. In both of them, all that concerns the eyes is pointedly brought out, yet both contain much that seems irrelevant in this place. This criticism is still more applicable to the interesting papers on tremors, reflexes, and gaits by Dr. Sailer, and to that on degeneracy by Dr. Walton. Just because a certain proportion of psychopathic patients have headache not referable to eye-strain and implying morbid sensitiveness, it seems going a little far to enter upon the whole subject of degeneracy and its physical marks, as a means of enabling the physician to diagnosticate his cases justly.

One more criticism of a general nature may be made—although it is not more applicable to this book than to most text-books, not indeed so much—and this is, that in a number of the articles the subject of treatment gets an insufficient notice. Truly enough, this is a topic which the physician often finds but little pleasure in discussing when incurable disorders are in question, and yet it is one that most readers turn to with great eagerness. And when it is seen what an immense amount of patient research is expended on the elucidation of some fine point in pathology, and how the exact anatomical findings of one case after another are described, it is impossible to help wondering whether similar amounts of searching toil expended on the study of the occasional successes in medical or surgical therapeutics might not be well repaid.

With these qualifications, we feel amply justified in giving hearty praise to the twenty-one writers by whom the twenty-three chapters, which constitute the work, were contributed. The subjects taken up cover the anatomy and pathology of the nervous system so far as the visual apparatus is concerned; the examination of the muscles of the eye; the affections of the related nerves; and the many diseases of the nervous and other systems in which visual troubles play a part. Dr. Posey also contributes an excellent paper on mental symptoms due to operations on the eyes, and Dr. Charles H. Frazier another on the surgical treatment of intracranial lesions related, even remotely—very remotely—to disturbances of vision.

It is difficult to comment on all these articles, but we mention as deserving of especial praise the various papers dealing with the mode of examination of the eye; the long, thorough, and judicious discussion of visual psychoses and neuroses, by Dr. G. E. de Schweinitz; the chapter, containing much that is rarely seen, on ocular disorders as related to the insanities, by Dr. F. X. Dercum; and that on amaurotic family idiocy and infantile cerebral palsies, by Dr. B. Sachs.

Dr. Spiller's anatomical observations are, as usual, admirable, and it is a tribute to him that several of the plates used in other articles are signed with his name. Dr. Mills' paper on the psychology of vision and the diseases of the cortex gives a very conscientious and careful discussion of certain aspects of these subjects, but we bow more to his anatomical and clinical accuracy than to his psychological insight. In fact of real psychology, at all comparable in value to his anatomy, we find but little.

The book is excellently printed and is well illustrated, if not with the great fulness that characterizes the masterly volumes by Wilbrand and Sänger.

J. J. P.

Atlas and Text-book of Human Anatomy. By Professor J. Sobotta, of Würzburg. Edited, with additions, by J. Playfair McMurrich, A.M., Ph.D., Professor of Anatomy in the University of Michigan, Ann Arbor. Volume I, pp. 258. Philadelphia and London: W. B. Saunders Co., 1906.

Manual of Anatomy, Systematic and Practical, Including Embryology. By A. M. Buchanan, M.A., M.D., C.M., F.R.P.S. (Glas.), Professor of Anatomy in Anderson's College, Glasgow, Scotland. Vol. I, pp. 596. Chicago: W. T. Keener & Co., 1906.

Volume I of Sobotta's *Atlas and Text-book of Human Anatomy*, which is now given in its English dress, comprises a description of the bones, ligaments, joints, and muscles. The descriptions are thorough, accurate, and refreshing in their conciseness—a feature that will be highly prized by students and practitioners, for whom, indeed, especially the book has been prepared. What is known as the Basel nomenclature has been adopted throughout, but the American editor has anglicized most of the terms, except those in the section on myology, and in those relations in which the Basel term is unfamiliar to English-speaking students he has added also the more familiar term. Excellent, however, as is the text, it seems, as it were, overshadowed by the illustrations—320 in number. Photographs were utilized as the basis of most of the drawings; the finished plates comprise multicolored lithographs, prints made by the so-called three-color process, colored half-tones, a few simple line etchings, etc. In fidelity to nature, wealth of detail, and artistic beauty the illustrations are not equalled by those of any similar book with which we are acquainted. There is little doubt that among the many atlases and text-books of human anatomy this deservedly must take a front rank.

Buchanan's book, although a manual, is somewhat comprehensive in its inclusiveness, being designed as a text-book of systematic anatomy and a manual of practical anatomy, or dissection, and including also brief notes on embryology. Volume I deals with osteology and the anatomy of the upper and the lower limbs. The text, like that of Sobotta's and most other anatomies, does not make very exciting reading; it is accurate and concise, but it does not differ from that of others in any noteworthy respect to call for particular comment—which in itself is praiseworthy, since there are many good anatomies. Almost one-half of the work is devoted to osteology—the best section of the book. The anatomy of the limbs is described as the structures are encountered in dissection. The guide to the dissection of the extremities appended to the description of their structures is rather inadequate; its utility would have been much enhanced by more precise directions relating to how to dissect, as well as what to dissect. The illustrations—268—are much less pretentious than Sobotta's. All are line cuts; most of them are original; many of them are colored—to indicate vessels, nerves, attachment of muscles, etc., and they serve their purpose well. The book is a personal volume, reflecting the views of a distinguished teacher, and doubtless it will be well received and prized by the author's students and friends; but, lacking a distinguishing feature, such, for instance, as the illustrations of Sobotta's book, it probably will not appeal to a very large circle of readers.

A. K.

PROGRESSIVE MEDICINE. A QUARTERLY DIGEST OF ADVANCES, DISCOVERIES, AND IMPROVEMENTS IN THE MEDICAL AND SURGICAL SCIENCES. Edited by HOBART AMORY HARE, M.D., Professor of Therapeutics and Materia Medica, Jefferson Medical College; assisted by H. R. M. LANDIS, M.D., Demonstrator of Clinical Medicine, Jefferson Medical College, Philadelphia. Vol. IV, December, 1906. Pp. 349. Philadelphia and New York: Lea Brothers & Co., 1906.

IN this, the concluding volume of the series for 1906, J. Dutton Steele devotes one hundred and twenty pages to diseases of the digestive tract and allied organs. Special attention is given to a discussion of the significance of occult blood in the diagnosis of gastric and intestinal ulceration, variations in gastric secretion, radiography in the diagnosis of gastrointestinal disease, the varieties and the treatment of constipation, the morbid physiology of visceral pain, cholelithiasis, the stools in disease of the pancreas, and the absorptive power of the peritoneum, with special reference to Schmidt's method of abdominal infusion. William T. Belfield discusses recent progress in diseases of the genitourinary organs, devoting special attention to tuberculosis, tumors, acute unilateral septic infarcts of the kidney, gonorrhœa, and prostatic hypertrophy. John Rose Bradford, discussing diseases of the kidneys, devotes especial attention to treatment. Recent progress in fractures, dislocations, amputations, and the surgery of the extremities is discussed by Joseph C. Bloodgood—one hundred pages. Bone tumors, tuberculosis, and Bier's hyperemia treatment are discussed in detail. H. R. M. Landis contributes a therapeutic referendum of sixty pages that will appeal to every general practitioner.

The volume is a worthy successor of those that have gone before; nothing of moment seems to have escaped the vigilance of the editors, and all that counts for progress has been included.

A. K.

CHEMISTRY: GENERAL, MEDICAL, AND PHARMACEUTICAL, INCLUDING THE CHEMISTRY OF THE U. S. PHARMACOPŒIA. A Manual of the Science of Chemistry and its Applications to Medicine and Pharmacy. By JOHN ATTFIELD, F.R.S., Professor of Practical Chemistry to the Pharmaceutical Society of Great Britain. Edited by LEONARD DOBBIN, Ph.D., Lecturer on Chemistry in the University of Edinburgh. Nineteenth edition, pp. 760. Philadelphia and New York: Lea Brothers & Co., 1906.

THE announcement of a nineteenth edition is, of itself, proof sufficient of unusual merit, and the additional fact that a book withstands the vicissitudes of forty years (since 1867) of scientific endeavor shows an extraordinary adaptability on the part of the author. As in earlier editions, so in the nineteenth, Professor Attfield and his editor, Dr. Dobbin, have successfully accomplished their purpose to produce a manual of chemistry in which not only the science of chemistry should be taught, but in which also the chemistry of every substance having interest for students of medicine and of pharmacy should be set forth in all attainable exactness. The new edition has been revised to conform to the new United States Pharmacopœia. One may regret that some reference to radium has not been included. Still, two generations of scientists have grown up and thriven on the book; doubtless it will serve as pabulum for at least a third. A. K.

DISEASES OF CHILDREN. A POCKET TEXT-BOOK. By GEORGE M. TUTTLE, M.D., Attending Physician to St. Luke's Hospital, St. Louis, Mo. Second edition, pp. 392. *Lea's Series of Pocket Text-books*. Edited by BERN B. GALLAUDET, M.D. Philadelphia and New York: Lea Brothers & Co., 1907.

THE excellence of Prof. Tuttle's work has carried it through two printings and now to a new edition, revised to represent advances to date. By considering only those diseases and phases of disease peculiar to the period from birth to adolescence, the author has found space to present all the essentials of pediatrics within a very compact volume. The various diseases are systematically discussed under appropriate subheadings, the typographical arrangement being such that any desired subject may be instantly found.

A. K.

PROGRESS

OF

MEDICAL SCIENCE.

MEDICINE.[1]

UNDER THE CHARGE OF

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Concerning Hemorrhages from the Kidney.—CASPER (*Berl. klin. Woch.*, 1906, xliii, 1271) states that the occurrence of sudden and often profuse hemorrhage from the kidney without any apparent lesion has attracted a great deal of attention the past few years. In many of the cases the usual causes of renal hemorrhage, stone, tuberculosis, tumor, kinking of the ureter, etc., can be definitely excluded, and the absence of any gross lesion has given rise to such terms as essential renal hemorrhage, angioneurotic hemorrhage, and renal hemophilia. Recently, however, a number of authors have opposed the view that bleeding can occur from a healthy kidney. In their own cases they were always able to find some lesion, and, although the lesion was often insignificant, they still look upon it as the cause of the hemorrhage. Casper reports four cases; all were adults in good health, and the occurrence of blood in the urine was the only symptom of disease. There was no albumin and no casts in the urine. Examination of the organ removed at operation revealed in the first two a well-marked, diffuse, chronic parenchymatous nephritis, demonstrating that such lesions may be present for years without albumin, casts, œdema, or circulatory disturbances and displaying as the only symptom repeated and protracted hematuria. In the other two instances most careful macroscopic and microscopic examination showed the removed kidney to be perfectly normal except for such slight and circumscribed changes as Casper thinks might be found in any kidney. These changes consisted in small areas of fresh infiltration and the circumscribed occurrence of fibrous tissue about a few glomeruli and small arteries; in the second case also general œdema of the organ. The œdema, Casper thinks, was the result and not the cause of the hemorrhage. The fresh infiltration could well be the result of handling the kidney during operation, and a little increase in the connective tissue about a glomerulus or small bloodvessel is such a common occurrence that we could not in reason attribute any great importance to its presence. He opposes the use of the term nephritis by Israel, Küster, and Rovsing to express the trivial changes they have found in the cases they report. To constitute a nephritis the changes must be bilateral and must be diffuse. In the examination of a large number of cases, by collecting the urine from two ureters separately, he has never found any evidence to support the view of a unilateral nephritis as described by Israel, Pausson, and Edebohls. Such trivial lesions he believes cannot, at least in his own cases, be held responsible for the hemorrhage. They are found so frequently in kidneys from which no hemorrhage has occurred that it is unjustifiable to settle upon them as a cause without more definite proof. In his cases there was every evidence,from the uniform distribution of the blood in the kidney, that the hemorrhage did not come from any one definite point. He thinks that, as far as our knowledge goes at present, we must accept that bleeding may occur from apparently healthy kidneys without any demonstrable lesion to account for the hemorrhage. He urges early operation in all cases, as without it a positive diagnosis is impossible and the early malignant tumor may be overlooked and, by the time a subsequent bleeding occurs, may have metastatized or become inoperable.

Agglutination in Pulmonary Tuberculosis.—Jessen (*Beit. zur Klin. der Tuber.*, 1906, vi, 209) has made agglutinating tests of the blood serum in a large number of sanatorium patients at Davos. His results fall into three divisions: (1) Patients who are treated by the usual sanatorium methods show, as a rule, a rise in their agglutinating power, and this rise is just as evident and just as high as in cases treated with tuberculin. Evidently the agglutinating power varies under natural conditions and is not, as has been claimed, a direct expression of a reaction to tuberculin injections. (2) Jessen finds that agglutination is of only slight value in prognosis. He divides his cases into three groups: (*a*) Cases that show marked improvement; some of these, originally severe cases, had gradually lost their symptoms and become a great deal better. Nearly all of these display a high agglutinating value, but some who had improved most favorably had a low value. (*b*) Cases clinically considered cured display in most instances a very low value, but a few a very high; and (*c*) cases which had run an unfavorable course showed neither a very low nor a very high value. Apparently, in most instances when improvement takes place the agglutinating power rises. In cases which run unfavorably there is little tendency for such a rise to occur. When improvement produces a cure, the value again tends to sink. Such a classification is accompanied by a great many exceptions, but still Jessen thinks the test is of some prognostic value. If in a given case the agglutinating value is high, the outlook is better than if under the same condition it were low. (3) As a diagnostic measure. In this relation he thinks only high dilutions should be employed. All his tuberculous cases gave values over 1 to 25. While lower values do not exclude tuberculosis, he thinks higher values suggest its presence. Jessen believes its special field in diagnosis is to lend aid in distinguishing between tuberculosis and other diseases when there is some evident pulmonary lesion present.

A Ring Test for Acetone.—LANGE (*Münch. med. Woch.*, 1906, liii, 1764). 15 c.c. of urine is mixed in a reagent glass with from 0.5 to 1 c.c. of acetic acid, and a few drops of a freshly prepared solution of sodium nitroprusside is added. Upon floating a small amount of ammonia upon this mixture an intense violet ring develops at the point of contact, if acetone is present. The test will detect acetone in a 1/400 per cent. solution. This is merely a convenient modification of the Le Noble reaction and has the advantage over Legal's test of not reacting to creatinin. Lange thinks the contact reaction gives a more distinct picture than the mass reaction. It has an advantage over the iodoform reaction in that alcohol and aldehyde do not give it. It is, however, not nearly so sensitive as the latter.

Typhoid Bacillus Carriers in an Insane Asylum.—NIETER and LIEFMANN (*Münch. med. Woch.*, 1906, liii, 1611). In an insane asylum with 900 female inmates, typhoid fever and dysentery have been endemic for many years, although no explosive epidemic attacks have occurred. Attempts to check the occurrence of the disease by isolation, careful attention to the water and food supply, and to the drainage have been of no avail. Being convinced that infection must originate within the institution itself, the stools of all the inmates were examined and in seven instances they were found to contain typhoid bacilli. Two of these patients were ill with dysentery and the two organisms were recovered from the stool. One patient agglutinated typhoid bacilli 1 to 100, and dysentery bacilli (Flexner) 1 to 800; the other 1 to 50 and 1 to 100 respectively. In the insane asylum at Hardte, the discovery and isolation of two typhoid bacilli carriers led to a rapid fall in the number of cases. Nieter and Liefmann hope to obtain the same results by the isolation of their seven patients.

Cultivation of Typhoid Bacilli from the Blood in a Bile-containing Medium.—CONRADI (*Münch. med. Woch.*, 1906, liii, 1654). It is now well known how important routine blood cultures are for the diagnosis of early and doubtful cases of typhoid fever. The organisms may be cultivated in a large percentage of cases and they occur with marked regularity in the first days of the disease and very frequently before the Widal reaction is positive. Conradi now reports a method by which he claims to be able to cultivate typhoid bacilli in practically every instance if the examination is made in the first week. As early as 1901 he called attention to the fact that bile prevents coagulation. It is also an excellent medium for typhoid bacilli to grow upon, probably acting favorably by neutralizing the bactericidal power of the extravascular blood. The medium as he now employs it consists of oxbile with 10 per cent. peptone and 10 per cent. glycerin. In a sterilized tube containing 10 c.c. of this medium from 2 to 3 c.c. of blood obtained from the ear or from a vein is introduced and the mixture incubated for sixteen hours. If typhoid bacilli are present they will have greatly increased in number and identification is readily made by transferring to the usual media.

Theoretical and Practical Consideration of Head's Zones in Visceral Disease.—Kast (*Berl. klin. Woch.*, 1906, xliii, 1033). Head has described certain areas of increased sensitiveness to pain and heat sensation in the skin corresponding definitely to disease of various viscera. These zones he has outlined to correspond with different spinal segments. They do not follow the distribution of the peripheral nerves. Their mode of origin he assumes is, so to speak, a psychical deception. We are devoid of pain sensation conveyed directly from the internal organs, but the sensory nerves from an organ come in close contact in the cord with sensory fibers running from the skin, and our subjective sensations may interpret impulses in the one as pain sensations in the other—referred pains. Kast is of the opinion that in many instances there is hypersensitiveness to touch impressions as well as to pain and thermal sensations. There is generated, however, in the segment a hypersensitiveness so that impulses which under ordinary circumstances are not painful are interpreted as such. This condition Kast calls a local neurasthenia. He points out, too, the importance of a general instability of the nervous centres in the development of the hyperesthetic areas. They are certainly better defined in neurasthenic patients. In a normal individual the continuous discharge of centripetal impulses from the internal organs run to the centres, but whatever changes they produce their effects remain in the subconscious sphere. Under conditions of increased sensitiveness to external and internal stimuli (neurasthenia) these impulses enter the conscious sphere and are frequently transformed into painful sensations. Lenander has shown that one may handle or insult in various ways the abdominal viscera without producing pain sensation; that pain sensation can be evolved only from the parietal peritoneum. His explanation of the hyperalgesic skin areas is the interstitial inflammation about and through the spinal nerves caused by a lymphangitis spreading from the diseased organ. Kast takes exception to such a view, urging that the skin zones do not follow the distribution of spinal nerves and that, if such a perineuritis existed, it would give rise to severe neuralgic pains. He believes, too, that under diseased conditions pain sensations do arise in the internal organs. As interesting as the discovery of Head's zones are, they have not proved of great practical value. In the first place, their occurrence is not constant. They are frequently present in gastric ulcer, particularly in anemic nervous patients and in instances with much pain. But in some of the most evident cases they are absent. The same is true of appendicitis and cholecystitis. In twelve cases of gastric carcinoma, all associated with a great deal of pain, Head's zones were present in only one. On the other hand, the zones occur in such a variety of conditions that no conclusion can be drawn, from their presence, of the nature of the disease. The localization, too, Kast finds practically unreliable. Different diseases in different areas may give the same zones. Still, it must be allowed that they frequently lend a valuable clue when a symptom is present, which may have, as a cause, one of a number of widely different sources, for instance, protracted vomiting, which may be cerebral in origin or come from a gastric ulcer or an appendicitis, etc., or attacks of indefinitely localized pain, which may arise from the heart, intestines, gastric ulcer, cholelithiasis, nephrolithiasis, etc. It is important in our investigations to exclude hyperalgesic areas arising from hysteria or from neuritis or cerebral or spinal lesions and the sensory disturbances which frequently occur with or after infectious diseases and in uremia. These usually have a different distribution. The difficulties in the way of uniform and reliable deductions being drawn from the mapping out of Head's zones are so great as to render the procedure of small practical value with our present means of investigation.

Myeloid Transformation.—Hirschfeld (*Berl. klin. Woch.*, 1906, xliii, 1064). Ehrlich, in his classical studies on the blood, drew a sharp line of distinction between the generation of the granular leukocytes and the lymphocytes, the former always arising in the bone-marrow, the latter always in the lymph glands and spleen. Extending these views to disease he drew the same distinguishing line between myelocytic and lymphocytic leukemia, the one type arising in the bone-marrow, the other in the glands and spleen. In the past few years, Hirschfeld, Kurpjuweit and others in man, and Dominici experimentally in animals, have demonstrated, in numerous instances of anemia and infectious diseases, the occurrence of myelocytes, neutrophiles, and nucleated red cells in the spleen and glands and occasionally in the liver. Kurpjuweit and Sternberg claim that myelocytes are regularly present in the spleen in small numbers. Hirschfeld cannot agree with this view, and he has, at his command, an extensive experience. The presence of granular cells he considers a true metaplasia, deriving them from preexisting lymphoid cells or the closely allied large mononuclear cells normally present in the spleen. Similarly certain myeloid tumors in the liver he believes to be autochthonous. In one instance he reports, where they were present in large number, he can see no other explanation of their occurrence, as there were at no time myelocytes in the circulating blood. In three cases of lymphatic leukemia, which he has recently observed and briefly reports in this article, he likewise found extensive myeloid transformation in the spleen and glands. The bone-marrow in three cases contained very few granular cells, while myelocytes and nucleated red cells were abundantly present in the myeloid areas of the spleen and lymph glands. The view that such areas may have been bone-marrow metastases he thinks is refuted by the fact that in two of the cases large numbers of myelocytes were never present in the blood, and in a third the myeloid transformation of the glands was extreme, while only slight in the spleen. It would be difficult to understand why metastases should have shown such a selective action for the glands. In the present state of our knowledge of blood histology, there can be no longer any objection to accepting mixed forms of leukemia. Cases of myeloid leukemia have been reported changing into a lymphoid type. Such evidence as Hirschfeld furnishes leaves no doubt of the occurrence of mixed types of the disease. In myeloid leukemia the spleen and gland tumors do not consist solely of myeloid tissue, but the lymphocytes have a large share in their structure. In the two forms of the disease one type predominates, but not at the expense of the other, which, likewise, hypertrophies. The sharp barriers established by

Ehrlich between the granular cells and the lymphocytes and between myeloid and lymphoid leukemia have been broken down. This does not mean that all distinction is lost and that myeloid and lymphoid leukemia become identical. They probably represent two different diseases which must be histologically and etiologically separated. But a large number of transition forms do occur.

SURGERY.

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An Experimental Study of Suture of Arteries, with a Description of a New Suture.—Dorrance (*Annals of Surgery*, 1906, xliv, 409) reviews the methods in use, with their advantages and disadvantages. He then describes his method and gives in detail his experiments on nine horses and five dogs. The method consisted in approximating intima to intima with a continuous through-and-through mattress suture modified by going back one-half a stitch every third suture, in order to overcome purse-stringing; over this he placed a continuous overhand suture. Special stress is laid on the following points: (1) Whatever suture is used the principle of placing intima to intima is absolutely essential, and (2) the suture and fringes of the intima should not be exposed to the blood stream.

Suprahyoid Pharyngotomy.—v. Hacker (*Zentralbl. f. Chir.*, Nov. 10, 1906) says that the technique of oblique or transverse pharyngotomy has been developed from observations made upon suicidal wounds occurring in this region, which, in general, lie chiefly below the hyoid bone. The view obtained of the entrance into the larynx and pharynx, through a suicidal wound, and the healing which followed, led to the development of subhyoid pharyngotomy as an operation. A similar suicidal wound above the hyoid bone suggested the performance of suprahyoid pharyngotomy. Hocher has recently, again, recommended very highly the subhyoid operation. He avoids tracheotomy when possible, performing it secondarily only when a laryngeal nerve has been wounded. When tracheotomy is not done he places the body in an oblique position. He does the operation under local anesthesia with applications of cocaine solution to the mucous membrane. v. Hacker found this method very satisfactory in two cases in which he did thyrotomy, or splitting of the thyroid cartilage, for the removal of cancers of the larynx.

It was Jeremitsch, of Moscow, who, in 1895, first recommended suprahyoid pharyngotomy in place of the subhyoid operation, in consequence of observations made in the treatment of a suicidal wound above the hyoid bone. The gaping wound gave a very free exposure of the entrance to the larynx, and of the posterior and upper part of the pharynx, and was closed with a primary suture.

The special advantages claimed for this operation are: the greater room and freer access afforded, especially when an operation is to be done on the root of the tongue or the whole of the tongue is to be extirpated; and above all it avoids the danger of wounding the laryngeal nerve, while the epiglottis is not in the least danger. v. Hacker confirmed these statements of Jeremitsch on the cadaver, and this led him to try the operation in the living when a suitable case presented itself. This was a round-cell sarcoma of the base of the tongue, in a man aged sixty-three years, who was very much emaciated and weakened. Because the patient desired narcosis it was given and was administered through a high tracheotomy wound, with the body in an oblique position and the head depressed. The incision was carried on each side to the edge of the sternomastoid. The lymph glands were easily removed, and the lingual artery ligated on each side beyond the cornu of the hyoid bone. After division of the muscles (mylohyoid, geniohyoid, and hyoglossus) access to the base of the tongue and, finally, to the posterior and upper part of the pharynx was very free. The rather large tumor, with a part of the epiglottis which was in the grasp of the tumor, was easily removed with the thermocautery. The remaining parts of the tongue were brought together throughout, the divided muscles were united, drainage was placed in the lateral angles of the wound, and the cannula removed. The healing of the wound was normal. Symptoms of heart weakness set in, however, and on the tenth day the patient died. He had been nourished through a tube in the nose. The autopsy showed degeneration of the heart, hypostatic pneumonia, and marasmus. The wound healed by first intention.

The suggestion is made that because of the free access to the posterior and upper part of the pharynx this operation should be done as a preliminary step in the removal of nasopharyngeal tumors. v. Hacker believes that it will prove to be a very advantageous operation.

Rupture of both Kidneys, with Intraperitoneal Hemorrhage; Removal of all of the Left Kidney and Part of the Right Kidney.—Franklin (*Amer. Jour. Surg.*, 1906, xx, 309) reports the following very interesting case: A girl, aged sixteen years, fell a height of four feet from a car, landing on her abdomen across a rail. She felt that she "had torn something loose on the inside," and, although she had some pain, she walked home, a distance of about a quarter of a mile. When seen by Franklin, about an hour later, she had a temperature of 98° and a pulse of 127, with other symptoms indicating severe shock. She vomited a greenish material several times. The abdomen was distended, the right rectus rigid, the right kidney region tender, with dulness on the right side, especially in the right iliac fossa (the patient was lying on her right side). By catheter two ounces of very bloody urine was withdrawn. Intraperitoneal rupture of the right kidney was diagnosed. Owing to objections on the part of the patient and her parents the operation was not begun until about eighteen hours after the accident. She stood the operation well for the first hour, but later did poorly because of the great loss of blood. A large quantity of blood and urine was found in the peritoneal cavity, which was cleaned out and flushed with normal salt solution. The right kidney was found low in the abdomen, lying directly in front and over the third and fourth lumbar vertebræ. It showed three transverse rents and was tied at its lower pole to the left kidney by a dense fibrous band a half-inch in diameter (horseshoe kidney?). The left kidney was literally torn into fragments, entirely without a capsule, and separated from the ureter. The vessels of the left kidney were ligated and all the pieces removed. A portion of the right kidney, which had been mashed into a pulp, was removed also. The remaining portion of the right kidney (not more than two-fifths of the original kidney substance) was in a very bad condition and showed two rents. It was packed with gauze. The abdomen was filled with normal saline solution after repeated flushings, then closed with through-and-through sutures of silkworm-gut, and drained. After-treatment for the shock was carried out. From the time the reaction set in, ten hours after the operation, the patient did well. The gauze was removed on the sixth day. A urinary sinus followed, through which most of the urine passed for the next five days. It closed on the sixteenth day. The patient sat up on the thirteenth day and left the hospital on the twenty-third day. She has enjoyed good health ever since (now more than six months) and passes a normal amount of urine.

Treatment of Tuberculous Abscesses.—Starr (*Brit. Med. Jour.*, 1906, ii, 923) says that abscesses following tuberculous bone disease, so long as they remain quiescent as closed sacs and do not become infected, add no danger element to the local or general condition. It is well known that a proportion of abscesses if properly treated disappear in the patient suffering with bone lesion. If the abscess is allowed to open spontaneously it will become infected very quickly with septic organisms. On the other hand, if it burrow too close to the skin, one of two things may happen: it may become infected with the organisms from the hair follicles or sweat glands, or the skin may become invaded with the tuberculous disease. As to treatment, aspiration has several advantages, but generally is not advisable. The injection of germicidal agents does not seem reasonable, as any agent which can be safely injected into a closed sac without endangering life can scarcely be expected to have any beneficial effect. Excision of the sac entire cannot be carried out in any of the abscesses from spinal disease, but is a perfect method for very small abscesses that are free of the joints in accessible parts of the extremities. Incision and drainage always result in a long-continued discharging sinus, often endangering the life of the patient and always making his condition annoying. Incision, evacuation of the contents, curetting of the wall, and closing of the wound constitute the treatment of choice. Of fifty-five cases so treated by Starr, seven afterward broke down, while the remaining forty-eight, so far as can be traced, have remained perfectly closed and aseptic for periods varying from four months to six years. Only five of the series required a second evacuation of contents, and one a third.

The Treatment of Wounds after Transplantation.—Weischer (*Zentralbl. f. Chir.*, June 23, 1906) objects to the open-wound treatment after Thiersch's skin grafting, because the discharge coagulates at the edges of the grafts and being retained underneath separates it. Nor does he believe with Isnardi that it is necessary to wait six weeks for the development of a proper granulating surface, in large traumatic defects of the skin surface. Only too frequently the back and posterior surfaces of the extremities are involved where it is difficult to relieve the pressure, and, therefore, to obtain a clean granulating surface. The wounds are frequently impregnated with dirt and oil. In relatively small wounds when the discharge is slight and serous, little difficulty in healing arises, whether the open method or the boric ointment and compression treatment are employed. When the secretion is abundant and purulent, very rarely can one obtain a wound that is entirely free from pus. The abundant streaks of pus which are usually present can be washed away with saline solution. The upper layers of the granulation tissue are then evenly removed with a proper knife, giving a smooth surface upon which to spread out the grafts. On a limb the part should be bloodless during the grafting, and when the Esmarch bandage is removed the hemorrhage should be controlled by the application of layers of lint moist with warm salt solution. Because the heat quickly disappears, warm water should be poured on almost continuously for about fifteen minutes or a half hour. Then the wound and grafts are covered with a layer of lint and that with cotton, both moist with the warm salt solution, and a bandage applied. When in bed the patient should have a wide mackintosh under the part and a wire frame over it to prevent contact of the bed-clothes with the dressings, and to favor evaporation of the discharge. Every two or three hours for two days, fresh, warm salt solution should be dropped on the dressing, until it is felt by the skin. This dressing favors the escape of the discharge from under each graft, prevents coagulation at the edges, retention under the grafts, and their separation from the granulating surface. The dressing is first renewed in forty-eight hours, after thoroughly soaking it. After two more days a boric ointment dressing is substituted. Weischel obtained a 100 per cent. of "takes" in three-quarters of the cases, 80 to 90 per cent. of "takes" in one-quarter, and did not completely fail in any.

High Hemorrhoids as a Cause of Occult Blood in the Stool.—Goldmann (*Zentralbl. f. Chir.*, June 30, 1906) says that from methodical examination of the rectum with a protoscope during recent years, he has several times found highly situated hemorrhoids to be the cause of obscure hemorrhages from the bowel. In one case about 10 cm. above the anus he found on the anterior surface of the rectum, small dilated prominent veins. From some, blood was oozing, while others were covered with clotted blood. The remaining mucous membrane in this region was the seat of a slight catarrh, but was otherwise normal. With a specially devised galvanocautery the veins were destroyed, through a proctoscope. A complete and permanent cure followed. Schreiber has localized solitary and multiple varices, 15, 16, and 19 cm. above the anus. He found venous anomalous conditions in not less than 10 per cent. of the cases he examined. The treatment of such hemorrhoids can only be operative, and they can be easily and permanently removed by the galvanocautery.

Appendicitis in Nursing Children.—Kirmisson and Guimbelott (*Revue de Chir.*, 1906, xxvi, 441) collected 26 cases under two years of age, 9 occurring in the first year and 17 in the second. Of the 26, 19 died and 7 were cured. Of the 9 cases occurring in the first year, all died. The 7 cases cured were all under eighteen months of age. Operation was performed in 19 cases, 7 in the first year and 12 in the second; 7 were cured and 5 died. From these facts Kirmisson and Guimbelott draw the following conclusions: Appendicitis in the first two years of life is not as rare as has been thought. Its evolution is rapid and its prognosis extremely grave. The only cases cured up to the present were those in which operation during the attack was performed; therefore, the indication to operate immediately seems clear. The greatest difficulty in reaching a decision to operate is the difficulty in making a diagnosis. Kirmisson and Guimbelott consider that it is, therefore, desirable that all cases of this kind be published.

Hemorrhage of the Scalp in Operations on the Skull.—Kredel (*Zentralbl. f. Chir.*, 1906, xxxiii, 1137) controls the always unpleasant and sometimes dangerous hemorrhage of the scalp in brain operations by a method which he considers simple and rapid. Before the incision is made, he applies two narrow metal plates to the scalp, by a ligature passed under the scalp along the bone, the plate and ligature on each side of the incision being parallel to the incision, and about 1.5 cm. from it. The plates may be from 5 to 7 cm. long, or shorter, and in case the longer are to be employed, it will be best to bring the needle out about the middle of the plate and to reintroduce for the rest of the distance covered by the plate. The two ends of the ligature are then caught in a notch at each end of the plate, and are tied and knotted over it, thus compressing the tissues of the included portion of the scalp between the ligature and the plate. According to the length of the incision, one or more plates may be used on each side of the incision. The plates are 1 cm. wide, 0.5 cm. thick, of variable length, and slightly curved to fit the vault of the skull. It may also be curved or straight to adapt itself to the shape of the scalp incision. These curves, however, should not be too marked, because the tightly drawn ligature tends to form a straight line between the two ends of the curved plate, so that the included scalp is not compressed properly. This can be prevented by passing a second smaller ligature around the plate and first ligature at their middle, to prevent them from sliding away from each other.

Bier's Treatment in Tuberculosis and Acute Infections.—Auffret (*Revue d'Orthopédie*, 1906, vii, 481) says that Bier applies his treatment to very diverse conditions, infectious and non-infectious, acute and chronic; but he embraces under it three different modes of treatment, all having the same object, congestion of the affected part. He thus includes hot baking, compression by the elastic bandage, and aspiration with cupping glasses or a special apparatus. Hot baking and the aspiration method are recommended in the non-infectious conditions; the former in chronic rheumatism, arthritis deformans, old arthrites, and chronic œdemas; the latter when one wishes to exercise a more energetic action (old traumatic lesions, etc.). The cupping apparatus of Klapp should be employed in the localized infections when pus has already formed, particularly in mammary abscesses and buboes, furuncles, lymphatic glandular abscesses, and superficial panaris. The anodyne effect of the elastic bandage upon the pain makes it the treatment of choice in gonorrhœal arthritis, because it soon permits immobilization. The anesthetic and bactericidal properties are of advantage in painful affections, as acute arthritis, early metastatic foci, and infected wounds. In the presence of grave suppuration (osteomyelitis, suppurative arthritis, deep phlegmons, panaris, etc.) the first indication is to make a free incision for the escape of the pus; but the venous stasis renders service of secondary importance. It aids in the formation of an œdema of which the bactericidal properties seem proved. It facilitates the escape of pus and permits in many cases the early stoppage of packing and drainage.

An Ideal Retention Bandage for Inguinal Hernia in the Infant.—FIEDLER (*Zentralbl. f. Chir.*, 1906, xxxiii, 1161) says that the non-operative treatment of this condition sometimes offers difficulties. Those methods in present use frequently fail, because they do not remain firm on these restless patients. The unavoidable soiling of the apparatus leads to eruptions of the skin, infection of the lymph glands, and pain from pressure of the apparatus. Fiedler improvises a supporting apparatus from a layer of white woollen yarn, of about twenty to thirty strands. He makes of these a loop about 35 to 45 cm. long, according to the size of the patient. The ends of these are fastened together by two pieces of white, slender, linen bands. The hernia is first reduced, the loop is passed around the body, and the ends, tied together by the linen bands, are passed through the loop. The point of crossing through the loop is made to lie over the hernial orifice, and the ends are drawn tightly around the thigh and fastened by the linen bands to the girdle about the abdomen. The hernia can be kept in place by the elastic pressure, while the apparatus is so cheap and easily made that it can be frequently changed and kept clean.

A Contribution to the Operative Technique of Orchidopexia.—DE BEULE (*Jour. de Chir., et Annal. de Soc. Belge de Chir.*, 1906, No. 8, p. 390) describes his method of operation as follows: After drawing down the testicle, he incises the skin of the fundus of the scrotum anteroposteriorly for about 3 cm. and brings the testicle out of the incision. A second incision of the same length in the axis of the limb is made in the thigh below the genitocrural crease, well within the femoral vessels and down to the deep fascia. The testicle is then sutured into the bottom of the thigh incision to the deep fascia, the outer edge of the scrotal incision having first been sutured to the corresponding edge of the thigh incision. The remaining corresponding edges of the scrotal and thigh incisions are then sutured together and the entire wound thus closed. The sutures holding the testicle to the deep fascia of the thigh pass through the tunicæ vaginalis and albuginea. A gauze and collodion dressing is laid over the circular line of sutures, which hermetically seals it. If the patient is a child, he is watched carefully for a few days, and his knees are tied together to prevent abduction of the thigh, and, therefore, tension on the wound. The sutures are removed in ten to twelve days, when the patient may get up and walk.

Later, the connection between the scrotum and thigh is divided, the testicle being carefully separated and the wounds in the scrotum and thigh closed. De Beule has obtained excellent results in two cases, which were far from being favorable.

THERAPEUTICS.

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The Serum Treatment of Exophthalmic Goitre.—G. A. WATERMAN (*Boston Med. and Surg. Journ.*, 1906, No. 7, p. 165) reports five instances of Graves' disease treated by means of hypodermic injections of serum derived from blood drawn from the jugular vein of a goat, two weeks or more after the removal of the thyroid gland. Two of the patients are considered as cured, two improved, and one unimproved. Waterman considers this number insufficient for estimating the value of the treatment, but, looking over all the instances in which serum-therapy has been employed, he feels that in this method we have a means which influences the disease favorably, and which may, with further modification, prove of more uniform value. In his opinion, it may be along this line or by the use of cytotoxic serum, which has been so successfully used by BEEBE and ROGERS (AMER. JOUR. MED. SCI., vol. cxxxii, No. 1, p. 149) that we are to find a specific means of relieving the distressing symptoms of this most troublesome disease. The doses of the serum employed were from 15 to 45 mm., given at first daily, the intervals being increased as benefit was obtained. Continuation of the treatment for months is necessary.

Serum in Exophthalmic Goitre.—S. P. BEEBE (*Jour. Amer. Med. Assoc.*, 1906, xlvii, 66) concludes a paper upon this subject as follows: "When all the conditions are considered, I believe it is fair to conclude that the serum is an agent of considerable value in the medical treatment of exophthalmic goitre. It has been used in all manner of cases in which it was possible to make a diagnosis, and in some cases in which the diagnosis was doubtful. Only a comparatively small percentage of trials has resulted in failure, a much larger number in improvement, while an encouraging number of individuals have been completely cured. In making up statistics, all the cases of which we have any record have been included. A number of the patients have begun treatment so recently that it is not possible to make any safe conclusion of the outcome. The number of instances of cure and improvement is so large that it cannot be ascribed to coincidence, and, under favorable conditions, I believe much can be accomplished by serum-therapy."

Prophylactic Antisepsis.—CREDE (*Münch. med. Woch.*, 1906, No. 24, p. 1160) states that the strictest asepsis in the management of accidental and operative wounds may fail to prevent primary or secondary infection and advises prophylactic antisepsis in the form of collargol. This agent is non-irritant and effective and is taken up by the tissue fluids and circulates through the body, without local caustic effect. In instances of fractures complicated with infected wounds sequels such as necrosis, lymphangitis, septic and phlegmonous processes may occur, no matter how thorough the cleaning of the parts has been. Here collargol assures quick healing without reaction. Tissues that seem to have lost their viability often regain vitality in a surprising fashion and recovery is usually rapid. Credé considers that every injury should be treated with this agent and that it is equally advantageous after operations, no matter how carefully conducted these may have been. Collargol may be employed as a 3 per cent. dusting powder mixed with finely pulverized lactose or one and a half ounces of a 1 per cent. solution may be used as an irrigation for wounds, cavities, the bladder, etc. Tablets of collargol may be inserted into bullet wounds, into the wound of a compound fracture, or may be placed upon contused brain tissue. Collargol may also be used for syringing, as a gargle, an eye wash, internally in wounds of the stomach, and in the form of bougies or suppositories. Argyria is said to never follow its employment.

The Serum Treatment of Experimental Cerebrospinal Meningitis.—SIMON FLEXNER (*Jour. Amer. Med. Assoc.*, 1906, No. 8, p. 560) describes certain experiments which he has made in the production of cerebrospinal meningitis in animals, and in the serum treatment of the same. He considers that the experiments may have some value in helping to understand the characteristics and effects of the Diplococcus intracellularis. The experiments concerning the prophylactic and therapeutic properties of antisera prepared from the diplococcus are sufficiently encouraging to warrant further study. How far the results obtained on guinea-pigs and monkeys can be applied in man, it is not safe to predict. Should further experimentation show that injections of antisera can favorably modify the experimental disease in monkeys, the case would not be without hope. Flexner does not think that the injection into the spinal canal of man of alien sera should be undertaken until their physiological action has been worked out in more detail in monkeys. If it were practicable to employ monkeys for producing the antisera to be used in man, the danger of serum intoxication might be lessened while the good effects of the immunity principles were retained.

Tetanus Antitoxin.—W. D'ESTE EMERY (*The Practitioner*, 1906, lxxvii, 478), in a paper upon serum-therapy, states that tetanus antitoxin is of little value in acute tetanus, but in chronic tetanus it is of great benefit in affording relief to the symptoms and in distinctly improving the chances of recovery. It frequently happens that the improvement which follows each injection is so marked and so greatly appreciated by the patient that it carries distinct conviction to the medical man. The dosage of the agent is simple; large doses are required and they should be repeated whenever their effect becomes apparently lessened, or, if there is no obvious effect, within a few hours. The best site of administration is not so well established. There are six possibilities: (1) Subcutaneous; (2) intravenous; (3) intracerebral; (4) intraspinal injection; (5) application to the nerve leading to the wound, and (6) local application. In chronic instances, subcutaneous injection will be adequate, though the advisability of intravenous injection may be considered. In very severe instances of acute tetanus, the intracerebral injection has given striking results; about 150 minims is injected into the lateral ventricle after drilling a hole or after trephining. Intraspinal injection is probably not more advantageous than hypodermic administration. Local application to the wound should not be relied upon, but may be employed in connection with subcutaneous injection.

Sodium Salicylate in Scarlatinal Arthritis.—Ralph Stockman (*Edin. Med. Jour.*, 1906, xx, 244) has followed a number of instances of scarlatinal arthritis and reports his observations as follows: In five of the patients no treatment was adopted and the symptoms subsided in two, three, four, seven, and eight days. To eleven patients, sodium salicylate was given in ordinary doses, and in these the joint symptoms subsided in one, one, two, three, five, six, seven, eleven, twelve, thirteen, and seventeen days. Thus, it would seem that the untreated patients ran the more favorable course. In most of the cases the arthritis was slight, recovery was apparently spontaneous, and it seemed immaterial whether the salicylate was given or not. This is in accord with clinical experience. In scarlatina, as in other microbic infections, the joint involvement is to be regarded as a part of the general poisoning; and, as sodium salicylate does not benefit the primary disease, it also leaves untouched the joint affection. While this is true in most instances, in others, as in the cases in which the joint implication disappeared in a day or two after the administration of the salicylate, it seems clear that we have to deal with a true rheumatic arthritis complicating the scarlatina. Stockman considers that only in rheumatic fever have the salicylates a specific action, and the joints are especially benefited because the remedy is secreted into them from the blood. He believes that the salicylates are as truly specific in acute rheumatism as is quinine in malaria, or mercury in syphilis.

The Action of Aspirin upon the Uterus.—F. Chidichimo (*Therapeut. Monatsschr.*, 1906, No. 8, 389) quotes the following statements of Dillberger, which are the conclusions of the latter drawn from his clinical observations: (1) Aspirin acts well in ovarian dysmenorrhœa, since it not only diminishes the general nervousness but also lessens the ovarian hyperesthesia. (2) In uterine dysmenorrhœa, with functional nervous disorders which have no organic basis, it acts as a strong analgesic. (3) In severe diseases of the adnexa and chronic metritis, the drug lessens the intensity of the dysmenorrhœa. (4) If one administers aspirin shortly before menstruation the dysmenorrhœa will be suppressed. (5) Aspirin has a beneficial influence upon amenorrhœa and menorrhagia. Chidichimo, himself, finds that aspirin slows uterine contractions and exercises an analgesic effect. It is not excreted in the milk of nursing women. It causes diminution of pain in the genital apparatus, whatever its cause may be. The usual dosage is 10 to 15 grains, two or three times daily. The drug is said to have no disturbing effect upon the stomach or kidneys nor does it cause headache or tinnitus.

The Specific Treatment of Tuberculosis.—H. HOCHHEIM (*Zentralbl. f. die Gesamte Therapie*, 1906, No. 8, p. 393) concludes a paper upon this subject with the following *resume:* In the treatment of phthisis we have no certain therapeutic agent; the treatment of the disease is tedious and requires much patience both in physician and patient. One of the most approved means of treatment is the administration of thiocol (potassium sulphoguaiacolate), which may be given in powder, capsules, or solution, in daily dosage of about 30 grains. The drug is tasteless and has no disturbing effect upon the stomach. Its particular disadvantage is its costliness.

The X-rays and Resorcinol in Lupus.—S. EHRMANN (*Sem. med.*, 1906, No. 37, p. 439) reports excellent results from the combination of these two agents. His technique is as follows: First, a dressing impregnated with a 33 per cent. resorcinol ointment is applied; this is removed after about twelve hours and the lesion is curetted with a sharp spoon, or a tampon wet with mercuric bichloride solution is applied. The procedure is repeated after four or five days, and *x*-ray treatment is begun. The resorcinol applications are continued at intervals. Later, dressings of boric-acid ointment are applied, and, after a time, the treatment above described is again instituted. The cycle is continued until cure is completed, which is usually accomplished in from two to seven months.

PEDIATRICS.

UNDER THE CHARGE OF

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The Influence of Vaccination upon the Evolution of Infectious Diseases.—L. JESIERSKI (*Jahr. f. Kinderheilk.*, 1906, xiv, 337) details some interesting observations upon this point, made possible by the occurrence of smallpox in a hospital ward containing scarlatina patients. A child aged eight years, suffering from scarlet fever, presented a smallpox eruption ten days after admission to the ward. In consequence, all of the children in the service were at once vaccinated; these embraced 22 scarlatinous, 10 tuberculous, 1 leprous, and 5 typhoid patients. Of the 22 scarlatinous, 11 were more or less severely disturbed by the engrafted vaccinia: with some the evolution of the scarlatina was prolonged, while with the others various complications were noted. None of these children had been previously vaccinated. In the remaining 11 patients, 6 of whom had been previously vaccinated, vaccinia seemed to produce no disturbance. Of the 10 tuberculous patients, 1 alone presented any abnormal phenomena. Among the 5 typhoids, all of whom were convalescing, 4 presented no unusual disturbance; in the fifth, however, there was a marked infiltration of the arm with a temperature rise to 102.6°, which became normal on the fifth day. In the leprous patient, a boy aged seven years, vaccination produced no complication. It is interesting to note that in the contents of the vaccinal pustule lepra bacilli were present.

Congenital Rachitis.—Marfan (*Semaine med.*, 1906, No. 41, p. 481) concludes a study of congenital rachitis with the following propositions:

1. There is a congenital rachitis latent at birth, or only to be detected by microscopic examination. In these cases the bony deformities are not appreciable until some time after birth. Whether this form can be distinguished from an early acquired rachitis by any definite sign is not known; but when a rachitis begins very early with craniotabes, and when the softening of the cranial bones is clearly appreciable before the fifth or sixth week, it seems probable that the disease began before birth.

2. A certain number of infants affected with congenital rachitis are born dead or non-viable. It is among these that cases of excessive osseous deformity are found; these are observed by the accoucheur during parturition and are often accompanied by visceral malformations. As pointed out by Porak and Durante, we now know that even in these cases of excessive deformity the clinical diagnosis is not always possible; that micromelia is not necessarily a distinctive feature of achondroplasia, but may exist also in periosteal dysplasia and in rachitis, though inconstant in the former and rare in the latter; and that it is sometimes necessary to employ the microscope and x-rays in order to distinguish these osseous dystrophies.

Two forms which especially interest the physician are those in which congenital rachitis, evident at birth, is compatible with life and continues to develop after birth, and in which the evolution demonstrates the nature of the osseous alterations observed at birth. Typical examples of these forms are furnished by the cases reported by Marfan.

3. In the one representing the rarest condition the skeletal deformities are from birth so extended and so characteristic that, though slight, one cannot mistake their nature: enlarged cranium, with prominent forehead and jutting bosses, enlarged fontanelles, and often separated sutures, distinctly enlarged costal nodosities, enlarged epiphyses, and curved diaphyses. If the child is carefully guarded and escapes serious infections the deformities show little increase, becoming slightly accentuated after birth, then remaining quiescent for a long time, and finally disappearing toward the end of the second year.

4. In the second form of the disease there is at birth craniotabes, either alone or associated with a slight costal rosary. Differing from the preceding form, the rachitis here progresses after birth, as if, at the time of parturition, it was in its first stage; later on one sees characteristic deformities of the cranium, thorax, and limbs develop. At the same time, as in the preceding case, if the child is well cared for and escapes serious infections, the rachitis remains quite slight in degree and disappears without leaving decided deformity. As common characteristics of these two forms, Marfan states that such children are born most frequently before term, are below normal weight at birth, and present, in the course of the first two years of life, an anemia more or less profound.

Perforating Scarlatinal Angina.—GUINDESSE (*Semaine med.*, 1906, p. 138) has observed three cases of primary perforating angina in the course of scarlet fever, which presented some analogies with the perforations of the uvula lately described by Méry and Hallé.

In the first case, a girl aged fifteen months, admitted to the hospital on the fifth day of a grave scarlatina, there were already at the time of admission two perforations symmetrically situated in the anterior pillars, having the form of longitudinal slits about 0.5 cm. in length, with smooth edges of deeper color than the surrounding tissues. The child died, after eight days, of septicemia.

The second case was that of a boy aged five years, admitted to the hospital on the third day of a particularly benign attack of scarlatina. As the result of a necrotic process affecting the uvula and developing after admission, a complete perforation occurred about the end of the second week. There was little tendency to healing and the perforation remained, though somewhat diminished in extent, at the end of fifteen days, when the child was removed from the hospital. The third observation concerns a girl of eleven years, with whom the disease was complicated from the beginning with diphtheria, and the perforation observed later in the anterior pillar could perhaps be attributed to the latter disease; but since it persisted at the end of three weeks, when there were no traces of diphtheria remaining, it is likely that here again the perforation was primitive. Contrary to the observation of Méry and Hallé, perforating angina was not accompanied, in Guindesse's cases, either by a general reaction particularly marked, or by phenomena of local inflammatory reaction; the temperature was not greatly elevated, and there was no submaxillary adenopathy.

Guindesse does not consider perforative angina as indicating necessarily a severe prognosis; it may occur with mild infections as well as with the severer forms. Perforation, however, being the consequence of penetration of the streptococcus deep into the tissues, should be considered as one of the manifestations of a general streptococcic infection, and, as such, increases in a certain degree the gravity of the prognosis.

OBSTETRICS.

UNDER THE CHARGE OF

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Mitral Stenosis and Pregnancy.—French and Hicks (*Jour. Obst. and Gyn. Brit. Empire*, September, 1906) contribute an interesting paper upon this subject, comprising a study of over 300 cases. Of 300 consecutive patients 205 were married. In 66 per cent. no direct relation between pregnancy and heart failure could be proved. The average number of children to each mother was 4.5. In 28 per cent. there was a direct relation between childbearing and heart disease. In 25 the heart trouble began in pregnancy, while in 25 the first symptoms were observed in the puerperal state. In 7, it was impossible to determine whether the heart disease showed itself first, before, during, or after the birth of the child. As regards the result of mitral stenosis complicating childbirth, among those patients in whom the heart disease did not begin in pregnancy, 33 per cent. died in the hospital. Of those in whom heart disease began during pregnancy, 35 per cent. died in the hospital. Among 13 patients having mitral stenosis, who married but never became pregnant, 46 per cent. died in the hospital. While it is difficult to draw conclusions from hospital statistics, the mortality among married women having children and suffering from mitral stenosis, does not seem to be different from that of unmarried women, with the same heart lesion. In endeavoring to form a prognosis concerning the recovery or death of a pregnant patient suffering from mitral stenosis, great difficulty is experienced. After reviewing a considerable number of cases, the authors find that many patients suffering from mitral stenonis, and pregnant, did not develop symptoms during pregnancy sufficiently severe to send them to the hospital. The majority of these patients showed the same symptoms which non-pregnant patients with mitral stenosis presented. The treatment of mitral stenosis during pregnancy consisted in rest in bed and the administration of digitalis. Induction of labor was rarely necessary, and there was little postpartum hemorrhage. If the patient is able to be up and about without cardiac distress, it is best for her to do so. If cardiac failure threatens, she must remain absolutely quiet. There was no evidence in this series of cases that mitral stenosis tended to sterility. Among the complications most severe and fatal was fungating endocarditis. This was present in 23 per cent. of cases in which failure was not attributed to pregnancy. In 41 per cent. of those patients in whom heart disease began during pregnancy, this complication was present. In 39 per cent. of single patients, endocarditis was also found. It was not, however, present in any patient who, although married, had not been pregnant. In the whole series of fatal cases, endocarditis was found in 29 per cent.

The writers conclude that pregnancy may cause serious and even fatal heart failure in cases of mitral stenosis. They believe, however, that the dangers of pregnancy in these cases have been considerably overestimated, because based upon statistics taken from patients who did not report for treatment until heart failure had developed. They also conclude that comparatively few of these patients are sterile, and that among them abortion is not especially frequent. Labor is usually successful. Heart failure develops most often after successive pregnancies. The treatment of the condition is the same as in the non-pregnant. It is not always right to deny marriage to women who have mitral stenosis. The general condition of the patient, and her circumstances in life, should be taken into consideration.

Abdominal Hysterectomy for Inversion of the Uterus.—Barbour (*Jour. Obs. and Gyn. Brit. Empire,* October, 1906) reports an interesting case of inversion of the uterus, complicated by fatty degeneration. The patient was a multipara, delivered at full term two months before admission to the hospital. Labor was difficult and terminated by the use of forceps. There was considerable hemorrhage. After the delivery of the child the patient could not empty the bladder, and after getting up, was examined by a physician, who informed her that inversion had occurred. There was no hemorrhage. On admission to the hospital the pelvis was contracted. The uterus was completely inverted. Under pressure, partial replacement occurred, but could not be successfully completed. Complete inversion recurred when pressure ceased. A week later the abdomen was opened and the patient placed in the Trendelenberg position. A cup-shaped depression was visible in the floor of the pelvis, crossed by a transverse slit, into which the Fallopian tubes were drawn The posterior rim of the cup was incised. Pressure was then made on the inverted fundus through the vagina, and the uterus was replaced. It was found to be the seat of marked fatty degeneration and was accordingly removed. Supravaginal hysterectomy was done, and one ovary was removed with the uterus. The patient was discharged three weeks after operation. Microscopic examination of the uterine wall showed fatty degeneration of the muscle fibers, and between them extravasation of blood. This process affected especially the outer third of the muscular wall.

Spontaneous Inversion of the Uterus: Vaginal Hysterectomy.—Ferguson (*Jour. Obs. and Gyn. Brit. Empire,* October, 1906) reports the case of a primipara who had a slow and tedious labor. The occiput was posterior, and the child was easily delivered with forceps without rotation of the head. The placenta was delivered by suprapubic pressure, the uterus contracting and retracting satisfactorily. There was no abnormal hemorrhage, the patient making apparently a good recovery. She did not nurse the child. At the end of the third week she resumed her usual duties, feeling perfectly well. Three weeks after this she had a considerable red, vaginal discharge. At the end of six weeks this suddenly increased, and was supposed to be menstrual. The bleeding increased, and she consulted a physician, who found her suffering from inversion of the uterus. An endeavor was made, under ether, to replace the womb, but this failed, the patient having very free bleeding. Two months after confinement she was seen in consultation. Manipulation produced such hemorrhage that the vagina was firmly packed with iodoform gauze to arrest the bleeding, and to try the effect of continuous pressure. The patient's condition was so critical that she was at once removed to a hospital in considerable shock. As the hemorrhage was not entirely controlled, the patient was anesthetized and thoroughly examined. The inverted uterus was red, congested, and bled upon the lightest touch. The orifices of both Fallopian tubes could be seen. Efforts to reinvert the uterus produced persistent bleeding. An incision was then made into the posterior fornix opening into Douglas' cul-de-sac. The index finger of the left hand was passed into the inverted cup when the constriction ring was found very unyielding. The posterior uterine wall was incised, but even this was not successful. Vaginal hysterectomy was then performed, with clamps. The ovaries were removed with the uterus, and a small subperitoneal fibroid was found near the entrance of the inversion. The patient collapsed after operation, but was resuscitated, and made a good recovery. The case was interesting for the following reasons: The etiology was obscure. A partial inversion probably existed when the placenta was delivered. and this must gradually have gone on to complete inversion. At labor the patient received skilled attention by two experienced obstetricians. The puerperal period was carefully conducted, and under constant observation. The condition developed very gradually, hemorrhage being the only significant symptom. Treatment was exceedingly difficult, because manipulation excited copious bleeding. The uterine wall was so friable that it could readily have been ruptured, and hence, a repositor could not be used.

Fifty Cases of Labor Complicated by Organic Disease of the Heart.—INGRAM and LONGRIDGE (*Jour. Obst. and Gyn. Brit. Empire*, September, 1906) report 50 cases of labor with organic heart disease, from the records of Queen Charlotte's Hospital: In 11,319 patients, these 50 cases were found. Of them 4 (8 per cent.) terminated fatally. In 1 of these, following influenza, the patient had failing compensation with fever. She was delivered by forceps, but died the twenty-eighth day after. Endocarditis, with infarcts in the kidneys and spleen, were present. The second case was admitted in a critical condition, compensation having completely failed. As the child was dead, and forceps failed, it was delivered by craniotomy. The patient died in shock soon after delivery. In the third case the patient, a multipara, had rheumatism and mitral regurgitation. Compensation had failed. Labor was short and easy, but the patient perished ten minutes after delivery. In the fourth case, also of mitral regurgitation, the patient was delivered suddenly while in a cab. She survived labor, but died from dilatation of the heart on the nineteenth day after. Among the primiparæ in this series of cases, five infants were delivered stillborn. Among the multiparæ, five infants died, none of which was viable. In only six of these patients had there been previous miscarriage. In a number of them premature labor came on spontaneously, while in some it was induced. Most of these patients had mitral regurgitation. Twenty of the fifty had stenosis and regurgitation; six had stenosis only; and three had mitral and cardiac disease together. Before labor, compensation was good in 35 of these patients. It was poor in 8, and decidedly bad in 7; only 8 of them had considerable quantities of albumin in the urine; in 27 there was none. Of the total number, delivery took place without assistance in 68 per cent. Among the 50 there were 14 forceps operations. Labor was induced in 6; craniotomy was done in 2; version in one. There were 2 cases of contracted pelvis, 2 of twins, and 2 of adherent placenta. Somewhat more blood than normal was lost in these labors. In 17 cases the condition after delivery was good among the primiparæ; 21 of the multiparæ passed through labor without distress. There were 2 deaths late in the puerperal period, and 6 of the patients suffered from dyspnœa; 3 had symptoms of sapremia. Longridge adds a note upon blood-pressure in pregnancy from the examination of 142 primiparous patients. As pregnancy advanced from the fifth to the eighth month, the blood pressure rose from 120 mm. of mercury to 140 plus.

GYNECOLOGY.

UNDER THE CHARGE OF

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Abdominal versus Vaginal Hysterectomy.—Hartmann (*Zentralbl. f. Gynäkologie*, 1906, No. 35) thinks that there is no longer any indication for the vaginal operation, even in cases of acute suppuration of the adnexa. He lost only three out of 201 cases of laparotomy for diseased tubes and ovaries, and only one out of seventy-seven hysteromyomectomies. He removes the cancerous uterus by the abdominal route, but does not extirpate the lymph nodes.

Changes in the Adnexa after Extirpation of the Uterus.—Burckhard (*Ztschr. f. Geb. u. Gyn.*, 1906, Band lxiii, Heft 1) from experiments with rabbits found that in animals that were killed during the first year after operation the ovaries remained practically normal. Later a progressive atrophy was noted, doubtless due to circulatory disturbances. The tubes were usually unchanged. When a portion of the uterus was left it underwent cystic degeneration in over 50 per cent. of the cases, the wall being much thinned and the cavity containing an albuminous fluid, evidently secreted by the utricular glands.

Resistance of the Germinal Epithelium of the Ovary to Infection.—Wolfheim (*Monatsschr. f. Geb. u. Gyn.*, Band xxiv, Heft 1) conducted a series of experiments in animals in order to determine the resisting power of the surface epithelium of the ovary. As mechanical (corpuscular) irritants India ink was used in preference to other staining materials. When introduced into the peritoneal cavity, isolated cells of the germinal epithelium were found to be stained within six hours, but the particles were always arrested in this layer when the ovary was not injured. Hence the inference that the function of this epithelium is to protect the ovarian stroma from injury from without. Cultures of anthrax, streptococci, and staphylococci were employed for bacterial experimentation, but the results were entirely negative. It was interesting to note that particles of India ink were often found within leukocytes, especially in the lymphatics at the hilum, thus demonstrating the clinical fact that metastasis within the ovary takes place in this way, and not by direct extension from the surface. These results were obtained only when the ovary was perfectly intact; after the slightest traumatism there appeared to be no limit to the extension of the staining.

Decidual Cells in the Non-gravid Uterus.—Tawildarow (*Abs. of Thesis in Zentralbl. f. Gyn.*, No. 39, 1906) examined scrapings from 67 uteri, in 56 of which curettement was done for endometritis. He concludes that in simple glandular hyperplasia no cells are of a decidual type are found, but the reverse is true when the glandular and interstitial varieties combined. These cells with increased protoplasmic contents are to be distinguished from true decidual by their flatter nuclei, homogeneous protoplasm, and greater tendency to stain with eosin and picric acid. The changes observed in the endometrium in connection with fibroids and cancer of the cervix are the same as in simple interstitial and glandular hyperplasia, the latter being most common in fibroid uteri. Macroscopically decidual membrane may be mistaken for hyperplasia. Microscopically, the error is impossible, since instead of the dense masses of large cells, rich in protoplasm and containing swollen (blasenförmige) nuclei, and arranged in a compact layer, the hyperplastic endometrium shows only scattered cells of this type. The interglandular tissue of the endometrium is really embryonal tissue, composed of cells surrounded by processes of protoplasm, no fibrous tissue being present under normal conditions. The surface epithelium has no true submucous basis, nor do its glands possess a limiting capsule.

Artificial Distention of the Bladder in Gynecological Operations.—Frankenstein (*Beit. zur Geb. u. Gyn.*, 1906), Band x, Heft 2, recommends distention of the bladder with boric acid or saline solution previous to opening the abdomen in order to avoid subsequent retention, basing his deduction on 875 laparotomies, in 45.3 per cent. of which he was successful. Ischuria after Alexander's operation was also prevented in 20 per cent. of the cases, but in only 16.9 per cent. of his vaginal sections. This preliminary distention of the bladder is said to allow the operator to enucleate adherent tumors more easily.

Sarcoma of the Vagina in Children.—Rollin (*Rev. Gyn. et de Chir. Abdom.*, 1906, tome x, No. 1) adds a case to 11 already reported. Clinically the neoplasm assumes the form of a cluster of grapes (*traubiges sarcom*), preceded by a stage at which it appears as a nodule on the anterior or lateral vaginal wall. The vagina is eventually filled with a reddish-brown mass which protrudes from the vulva, and extends upward to the portio and cervical canal, rarely to the body of the uterus. The neoplasm has a marked tendency to break down and to form metastases, and the bladder is usually involved, cystitis and pyelonephritis being common complications. The rectum is rarely involved.

OTOLOGY.

UNDER THE CHARGE OF

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Some Points in the Diagnosis of the Complications of Temporal-bone Disease Based upon a Study of 135 Fatal Cases.—A. L. WHITEHEAD (*Archives of Otology*, 1906, xxxv, No. 5). During the last fifteen years, 892 cases of temporal-bone disease have been operated upon at the General Infirmary at Leeds, by Whitehead, his colleagues, and his predecessors. 146 patients died. A study of these fatal cases, in which careful postmortems revealed lesions in some cases unsuspected during life, or in which the symptoms rendered a correct diagnosis a matter of the greatest difficulty, presents many interesting features.

Of the 146 patients, 11 died from other causes, such as diabetes and cardiac disease. In the remaining 135 patients, death was more or less directly referable to the temporal-bone disease. 11 patients died from exhaustion; 10 of these were wasted infants under one year of age; all had very extensive mastoid disease and subnormal temperature after operation, and died of inanition in from ten to thirty days afterward. In young children with defective stamina, it is certainly wiser not to aim at complete removal of all disease at one operation; the establishment of good drainage and the removal of the more serious disease should be accomplished at the first stage, and at a later period, a more complete and satisfactory radical operation performed.

In 22 cases, acute miliary tuberculosis caused a fatal issue; in 18, the meninges were affected and the symptoms those of tuberculous meningitis. In 8 of the 22 cases some other primary forms were present, but in the remaining 14 the temporal-bone disease seemed to be the sole focus of infection; 19 patients were under two years of age; in every case either symptoms of tuberculous meningitis or distinct evidence of general tuberculosis were present before operation, and in no case could the operation be regarded as the originating cause of a general disseminated miliary tuberculosis. In 5 patients when admitted an ordinary catarrhal pneumonia complicated an acute mastoid disease following upon chronic otorrhœa, the pneumonia proving fatal. In 3 cases an extradural abscess, situated over the sigmoid sinus, was evacuated during the mastoid operation, death subsequently ensuing from septic bronchopneumonia. In these cases rigors and remittent temperature had preceded operation, but the sinus was found to contain fluid blood. No septic thrombus or alteration in the vessel wall was found postmortem, although the infection must clearly have been conveyed through the sinus. No uncomplicated case of extradural abscess resulted fatally.

Of the remaining 93 cases, serous meningitis was present in 1, general meningitis in 33, meningitis and sigmoid sinus thrombosis in 10, meningitis and cerebellar abscess in 5, meningitis and temporosphenoidal abscess in 6, sigmoid sinus thrombosis alone in 1, with pneumonia in 9, with cerebellar abscess in 7, temporosphenoidal abscess and thrombosis of the petrosal sinus in 1, temporosphenoidal abscess alone in 8, temporosphenoidal abscess and cerebellar abscess in 2, and cerebellar abscess alone in 10.

In the 17 cases of temporosphenoidal abscess, intense headache was present in all, vomiting in 13, drowsiness deepening to coma in 10, optic neuritis in 5.

In the 9 uncomplicated cases, temperature and pulse were subnormal in 2, in the cases with meningitis the rate was much above normal, and in the 2 cases with cerebellar abscess subnormal. In the 6 cases with meningitis, delirium and restlessness were present. In only 1 case, and that one of the uncomplicated ones, was the pupil on the affected side dilated and fixed. In 4 of the 22 cases the patellar reflexes could not be elicited. Sudden death occurred in 2 of the uncomplicated cases. In no case was an acute mastoid abscess present. One of the cases with meningitis had rigors and developed a septic bronchopneumonia. Among the 21 cases of cerebellar abscess, headache was present in 19, vertigo in 5, vomiting in 13, drowsiness in 11, optic neuritis in 3, subnormal temperature and slow pulse in 3, constipation in 4. Rigors occurred twice in the cases with meningitis and twice in those with latera sinus thrombosis. In the group with meningitis, delirium, restlessness, and muscular twitchings were a conspicuous feature. Sudden death occurred in 4 of the 10 uncomplicated cases, and in 1 other respiration ceased during the operation, and, although the heart was kept going for several hours, spontaneous breathing was never re-established. In 10 of the 21 cases an external mastoid abscess was present. Pupils were unequal in 1, nystagmus present in 3. The patellar reflexes never exaggerated, in 2 cases not obtainable. Of the 20 cases of sigmoid sinus thrombosis rigors were present in 14, high and often remittent temperature in all, headache in 7, optic neuritis in 5, vomiting in 9. Of the 33 cases of meningitis, headache was present in 21, vomiting in 12, mental excitement and delirium in 16, drowsiness in 9, muscular twitchings in 12, convulsions in 10, pupils dilated in 2, unequal in 3, optic neuritis in 8, elevated and fluctuating temperature in 21. Increased patellar reflex in 2, absent in 3; conjugate deviation of the eyes from the affected side in 3, toward the affected side in 2. External mastoid abscess was present in 17. The absence of the classical symptoms was a striking feature of these cases, but the author especially mentions the fact that this was not due to omission to note symptoms; in nearly all the cases definite mention having been made of the absence of symptoms of intracranial complications when they might have been expected. Headache was almost uniformly present in those cases in which a brain abscess was present. Vomiting was very frequently noted, but was absent in half the cases of uncomplicated cerebellar abscess. Drowsiness or coma was about equally frequent in cerebral and cerebellar abscess. Optic neuritis was more frequently observed in cerebral than in cerebellar abscess, and was rather remarkably common in the cases of meningitis, being present in eight out of thirty-three. Subnormal temperature and slow pulse were comparatively infrequent, being found in only 8 cases of abscess out of 38. Infection of the sigmoid sinus and meningitis were almost invariably associated with high temperature, and in the former group the usual remittent temperature was found in all but 2 cases. The condition of the pupils was rarely an aid to diagnosis; in only one case of temporosphenoidal abscess was the typical dilated pupil on the side of the lesion present. Conjugate deviation of the eye was not present in any of the cases of cerebellar abscess, although found in 1 case of cerebral abscess with meningitis, and in 5 cases of simple meningitis. Nystagmus occurred in 3 cases of cerebellar abscess. The patellar reflexes were absent in 4 cases of cerebral abscess and in 2 of cerebellar. Delirium, muscular twitchings, contractions, or convulsions were almost uniformly present in all the cases in which meningitis caused death. Rigors occurred in 16 out of the 26 cases of thrombosis of the sigmoid sinus, but were never observed in any uncomplicated case of cerebral or cerebellar abscess, and in only 1 case of meningitis. There was an antecedent history of chronic otorrhœa in 21 out of the 33 cases of meningitis; in 17 out of the 20 cases of sinus thrombosis; in 20 out of 21 cases of cerebellar abscess; in 13 out of the 15 cases of cerebellar abscess, and in 1 out of the 2 cases of combined cerebral and cerebellar abscess. External mastoid signs were never observed in association with temporosphenoidal abscess, but occurred in about half the cases in which cerebellar abscess or meningitis caused death. Examinations of the cerebrospinal fluid obtained through a lumbar puncture, and of the blood, were made in a number of instances, but were never found to be of diagnostic significance in the early stages of those cases in which the diagnosis was uncertain. Positive indications were only obtained when the diagnosis was beyond doubt.

PATHOLOGY AND BACTERIOLOGY.

UNDER THE CHARGE OF

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Disease of the Arteries in the Course of Acute Infections.—Compared to the care which has been given to the study of the heart muscle in acute infectious diseases, the changes in the peripheral arteries, except for a few scattered observations, have been practically neglected. The subject has been dealt with most satisfactorily by Simnitzky, who has recently discussed the question of arterial sclerosis in the young. In a previous communication WIESEL has described degenerative changes in the peripheral vessels in typhoid fever, which are encountered more frequently and appear more extensive than the lesions in the myocardium. In a second communication from Kretz's laboratory (*Ztschr.*

f. Heilkunde, 1906, xxvii, 262) Wiesel describes the changes which he has observed in the peripheral vessels of young people, and even children and infants, dying from various acute infectious diseases. All parts of the peripheral arterial vascular system were examined. Eighty cases of diphtheria were studied, eighty of scarlet fever, and many cases of other infectious diseases. In all cases he found constantly a disease of the peripheral vessels that manifests itself as a degeneration of the smooth musculature and of the elastic fibers, which is to be seen first in the middle coat and in the majority of cases is confined to this one tunic. The degeneration appears in localized areas which in certain vessels may be very numerous, and in severe cases leads to a veritable necrosis of the vessel wall. These necroses heal either through scarring or by *restitutio ad integrum.* In many cases, however, the process extends into the intima, in which case this coat undergoes sclerotic changes, whereby permanent injury is produced. Though these changes are usually of microscopic nature, in extreme cases they may be visible to the naked eye. The disease affects all arterial vessels down to those of the caliber of the digital arteries. The exact localization varies in different cases, but the vessels most constantly diseased are the aorta, the cerebral and coronary arteries. In general, the lesions may be classed in two groups: those in which the elastic fibers are involved principally, and those in which the musculature receives the brunt of the injury. Lesions of the first type are found in diphtheria, typhoid fever, influenza, and pneumonia, and Wiesel considers that they are produced by bacterial poisons; the second variety of lesion is seen in scarlet fever and the septic pyemic diseases.

The Bacterial Contents of the Nose in Infectious Diseases, with Special Reference to Epidemic Cerebrospinal Meningitis.—HASSLAUER (*Centr. f. Bakt.*, orig., 1906, xxxxi, 796) has investigated the nasal flora in healthy persons, exposed and unexposed to infectious disease, and in persons suffering from epidemic cerebrospinal meningitis and other infectious diseases. He believes that most organisms which enter the nose with the air current are arrested in the anterior parts, where they may be overgrown by saprophytes, and usually cause no harm. A primary or secondary rhinitis may occur in the course of acute infections which is quite analogous to middle-ear disease occurring in these conditions. Hasslauer isolated the meningococcus from the posterior nares of patients suffering from epidemic meningitis. A rhinitis may or may not be present in these cases. The organism was obtained from apparently healthy mucosa. Cultural and agglutination methods were used for the identification of the organism, and the author emphasizes the importance of these procedures for the accurate diagnosis of the meningococcus.

The meningococcus was not found in twenty persons unexposed to the disease, but it was isolated from nine apparently healthy persons who had been in close contact with meningitic patients. The presence of the organism in some of these cases produced no apparent ill effects, but Hasslauer believes such individuals are important factors in spreading the disease. He concludes that the nose is the portal of entry for the meningococcus, and considers it likely that it is carried from the nose to the brain by the blood stream. As argument for this he cites the fact that the organism is not infrequently found in the circulating blood, especially early in the disease. He was able to isolate it from the circulation four times. After treating the healthy meningococcus carriers with an antiseptic powder used as snuff, no cases of meningococcus arose from them, as far as could be traced.

A Study of the So-called Infectious Lymphosarcoma of Dogs.—Beebe and Ewing (*Jour. Med. Research*, 1906, xv, 209) have made an extensive study of this interesting malignant tumor which occurs in dogs, and is transmitted from one animal to the other through coitus. The primary growths are found on the genitals of dogs, and metastases may occur in many organs of the body. The tumor is, however, not especially malignant. Microscopically it closely resembles large-cell lymphosarcoma of man. The cells resemble the large proliferating cells of lymph follicles, but are polygonal more often than round, not arranged in alveoli, and are supported by thin strands of connective tissue. Beebe and Ewing do not attempt an exact histological classification of the tumor, but the general histological characteristics suggest to them the diagnosis of alveolar sarcoma or endothelioma. Many implantation experiments were performed. Small subcutaneous grafts and subcutaneous injections of suspensions of tumor cells were used. With one tumor all of the animals in which the first method was used developed typical tumors in three to five weeks. The second method gave negative results. The authors found that old dogs were more susceptible than young ones. Spontaneous cures resulted in some cases. They were unable to reinfect four animals in which recovery had taken place. The general health of the dogs seemed to have a rough relationship to their susceptibility. Recovery usually took place in vigorous well-nourished, young dogs. In multiple inoculations all tumors showed the same rapidity of growth or regression. The crucial point which Beebe and Ewing endeavor to settle is, whether, after grafting the tumor, the new-growth arises from the transplanted tissue, or whether its origin is from the surrounding tissue, as claimed by others. By making histological studies of transplanted tumors and the surrounding tissue at varying intervals after grafting, the authors conclude that although the central portions of the transplanted material usually dies, small islands of tumor tissue remain alive, from which the new tumor growths arise. Their experience leads them to agree with Sticker, that the infectious lymphosarcoma of dogs is a true malignant neoplasm and not a granuloma. They present no evidence of a parasitic cause for the tumor, and believe that if it is due to a parasite the parasite has so vital and intimate a relationship with the tumor cell that it is impossible by their experiments to make any distinction between them.

The Origin of Blood Platelets.—Though many explanations have been offered to account for the origin of blood platelets, none seems to have gained general acceptance, and Wright (*Virchow's Archiv.*, 1906, clxxix, 55), who has made an extensive comparative study of blood platelets in a large series of animals, considers that all the theories hitherto advanced are wrong. Wright's studies go to show that the blood platelets are derived from the protoplasm of the giant cells of the bone-marrow and spleen. These giant cells are the "megokaryocytes" of Howell. By a special technique he has been able to stain blood platelets in fixed tissue. The differentiation is eventually the same as that obtained with the modified Leishman-Romanowsky stains;

the central granular part of the platelet takes a red or violet stain, the peripheral hyaline portion a blue stain. With this stain the giant cells present a characteristic appearance. There is a central portion which forms the principal part of the cytoplasm, and is made up of small granules which stain red, and a narrow peripheral zone of hyaline protoplasm that stains blue. The gaint cells being amœboid throw out pseudopodia, which are composed chiefly of the outer hyaline cytoplasm, but show in the central part masses of the pink granules. The pseudopodia are sometimes seen projecting into bloodvessels. Pieces are pinched off the end of pseudopods, or whole pseudopodia may be seen lying free near a cell. These pseudopodia are, in their staining reactions, precisely like plates, while the pinched-off pieces cannot be told from platelets. By this pseudopod formation and pinching off process the entire protoplasm of a cell may be lost, leaving a naked nucleus lying in the midst of the cell remnants. Wright believes that the platelets are formed from the protoplasm of the giant cells in this manner. To uphold this theory Wright gives further arguments in its favor. Thus blood platelets occur only in mammals, animals which have giant cells in their bone-marrows. Wright has demonstrated platelets in all mammals of the different orders except the opossum and the camel, and except in the opossum has found giant cells in the marrows of all mammals examined. The so-called spindle cells of the birds, amphibia, and reptiles are analogous, not to the platelets, but to the giant cells of mammals. Further, the blood platelets appear in the blood of the embryo at the time when the giant cells appear in the marrow. The results of blood-platelet counts in the human being in various diseases go to show that the platelets are increased when the giant cells of the marrow are increased, and *vice versa*.

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THE

AMERICAN JOURNAL

OF THE MEDICAL SCIENCES.

MARCH, 1907.

ORIGINAL ARTICLES.

ACUTE DILATATION OF THE STOMACH, AND ITS RELATION TO MESENTERIC OBSTRUCTION OF THE DUODENUM.

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Although in recent years contributions to the subject of acute dilatation of the stomach have been fairly numerous in England and Germany, in this country the subject has received but little attention. Nevertheless the condition is one of considerable clinical interest and importance. Cases are encountered by the surgeon, the gynecologist and the orthopedist, as well as by the physician, and the condition has developed during apparently normal convalescence from operations or from some severe illness, changing, within a few hours, perhaps, the whole aspect of the case. While in most recorded instances the disease has progressed rapidly to a fatal termination, evidence is accumulating to show that in many cases life may be saved by timely recognition of the condition and by suitable treatment.

The possibility of such acute dilatation has long been known. Brinton, writing in 1859, discusses the subject and gives a short account of its clinical features. It was Hilton Fagge, however, who first directed general attention to the condition and gave an accurate description of the characteristic symptoms and physical signs. Since that time (1873) cases have gradually accumulated, until in 1902 Campbell Thomson was able to collect 44 instances of the disease—most of them with autopsy reports. The more recent review of the subject by Neck was based upon 60 cases. For the present study of the condition 102 cases have been collected and analyzed.

The accumulation of cases has made it evident that, in many instances at least, the clinical picture is very distinct and easily recognizable, and that the diagnosis is often not difficult if the possibility of the disease is borne in mind. There is also reason to believe that with the earlier recognition of the condition it will be possible to treat successfully many cases which have heretofore proved fatal. Concerning the pathogenesis of the disease, however, our knowledge is by no means so satisfactory and there exist still much confusion and uncertainty as to the nature of the affection and its causation.

In studying the recorded cases it soon becomes apparent that the condition of acute dilatation of the stomach rests upon no single pathological basis. A consideration of the extremely diverse etiological features and of the varying postmortem findings makes this very evident. Nevertheless it seems to have been clearly demonstrated within the last few years that a very important cause—perhaps the most important single cause—of such stomach dilatation is the sudden incarceration of the lower end of the duodenum between the root of the mesentery which passes in front of it and the vertebral column behind. The possibility of such mesenteric obstruction of the duodenum was expressly noted by Rokitansky in 1863. No illustrative cases were recorded, however, until 1891, when Kundrat, under the caption "Concerning a Rare Form of Intestinal Incarceration," described three such cases. These all had run a rapidly fatal course, and in two of them the necropsy showed great dilatation of the stomach as well as of the duodenum. Schnitzler, in 1895, reported another fatal case with enormous dilatation of the stomach and duodenum, and also an apparently similar case cured by treatment directed to the relief of the supposed duodenal compression.

The subject received no other notice, however, until Albrecht, in 1899, published a very complete and careful study of it. He reported two cases of acute gastric dilatation with the same mesenteric constriction of the duodenum, and discussed at length the relation of the two conditions to each other. Since Albrecht's communication a good deal further study has been made, chiefly in Germany, and a number of additional cases have been recorded. It should be mentioned that in 1900 Robinson, of Chicago, without knowledge of the literature of the subject, and believing that he was describing a hitherto unrecognized condition called attention to the possibility of such compression of the duodenum by the root of the mesentery and reported a fatal case associated, as usual, with enormous dilatation of the stomach.

The following similar case occurred in my service at the New York Hospital, and through the kindness of Dr. Farr of the house staff I was fortunate enough to secure photographs of the postmortem appearances.

Male, aged twenty-one years, Italian tailor, was admitted to the hospital on August 1, 1905. He had had malaria ten years before in Italy, but otherwise had always been perfectly well except for an occasional slight cough. There was no history of tuberculosis in the family. For four weeks before admission he had noticed progressive weakness and loss of flesh, had had slight cough, some dyspnœa, and some profuse night sweats. No pain. During the last few days he had been irrational at times. The bowels were constipated. Admission: temperature, 99.8°; respiration, 36; pulse, 116.

Physical Examination. Man of medium frame, very poorly nourished; mucous membranes pale. Pupils of equal size, reaction normal. Tongue moderately coated. Throat congested and dry. Neck somewhat stiff. No subcutaneous œdema. Lymph nodes in neck, axillæ, and groins palpable.

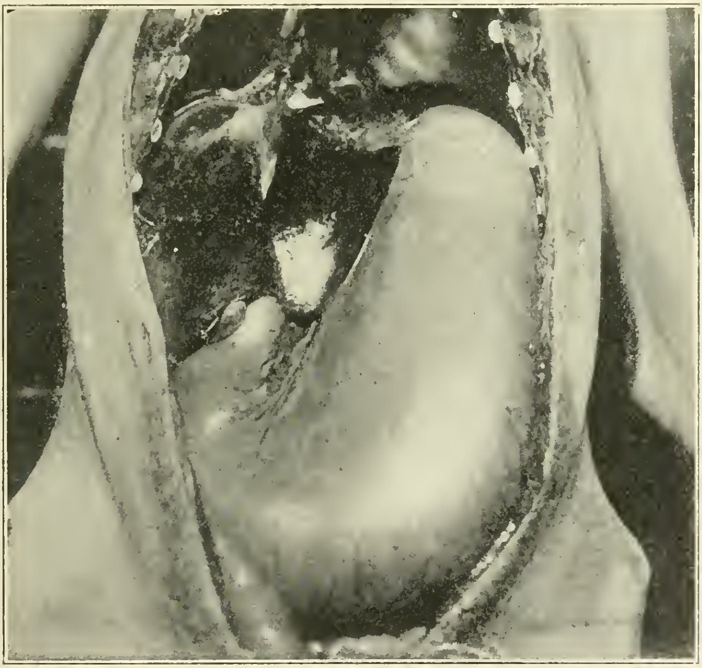

FIG. 1.—The dilated stomach due to mesenteric obstruction of the duodenum.

Lungs. In front slight dulness at right apex. Exaggerated vocal fremitus over same area. Vesicular breathing everywhere. Sibilant and sonorous rhonchi over both chests. Some medium-sized moist rales, especially with expiration, in both axillæ. Behind, slightly impaired resonance at the right apex. Auscultation signs similar to those in front.

Heart. Maximum impulse in fourth space four and one-half inches to left of midsternal line; action feeble, regular, and rapid. Relative dulness begins above at third rib, on the right side at right border of sternum, on the left side at line of apex. Sounds all normal except some accentuation of second pulmonic. Pulse rapid, regular, small, and weak. Artery walls somewhat thickened.

Abdomen. Retracted and somewhat rigid, has rather a doughy feel, but no masses can be felt. No rose spots. Liver dulness reaches to two fingers' breadth below costal margin, edge indistinctly felt. Right kidney palpable. Edge of spleen felt at costal margin.

The temperature rose at once to 103° and thereafter ranged constantly between 100° and 104°, with morning remission and evening rise, forming a chart very suggestive of typhoid fever.

Repeated examinations of the sputum throughout the illness yielded negative results for tubercle bacilli. Tests for the Widal reaction were all negative. Leukocytes: August 2, 34,000 in c.mm.; 8th, 26,000; 21st, 12,000.

August 15. Hemoglobin, 56 per cent.; red cells, 4,000,000. Examination for malarial plasmodia negative. Blood cultures taken on two occasions failed to yield any growth. Urine showed at each examination a trace of albumin and a few hyaline and granular casts. Diazo reaction positive.

5th. Condition and physical signs almost unchanged since his admission. Fever continuous. Mental state "queer." Takes very little food.

9th. Small moist rales scattered irregularly throughout both chests anteriorly; edge of spleen now felt over finger breadth below costal margin.

14*th.* Still slight dulness at right apex in front. Auscultatory signs of general bronchitis.

23*d.* General condition worse. Involuntary micturition and defecation. Will take almost no food, but drinks water freely. Fed by gavage. Spleen is decidedly larger than on admission. Complains of no pain. Belly retracted and somewhat rigid. No rose spots. Diagnosis of acute miliary tuberculosis seems very probable by exclusion, but in the absence of evidence of ocal tuberculosis cannot be made with certainty.

28*th.* Patient has been eating better for past day or two. Slept soundly last night. This morning about 9.30 suddenly complained of severe abdominal pain. He was seen almost immediately by the house physician, who found a marked distention of the left side of abdomen, especially above the umbilicus. Lower border of this circumscribed prominence could be made out just below umbilicus. The distended area gave deep tympany on percussion, was very tender to pressure, but there was no real rigidity of the abdominal muscles. The severe pain continued, the general condition grew rapidly worse, and at 12.40 P.M. (three hours after onset of pain) he died. There was no vomiting at any time.

Autopsy. Two hours after death (Dr. Biggs).

Anatomical Diagnosis. Acute general miliary tuberculosis; chronic tuberculosis of the bronchial and mediastinal lymph nodes; enormous dilatation of the stomach; obstruction of the duodenum by the root of the mesentery.

Body greatly emaciated, slight rigor mortis. No subcutaneous œdema. Abdomen somewhat distended. Upon opening the abdomen an enormously enlarged stomach presents. Peritoneum normal. The pericardial sac contains 150 c.c. of clear serous fluid. Near the base of the right ventricle is a miliary tubercle in the visceral layer. The heart is small, the cavities slightly dilated, the muscle pale and soft, the valves and coronary arteries normal. The bronchi contain much mucopus. The lungs are of light

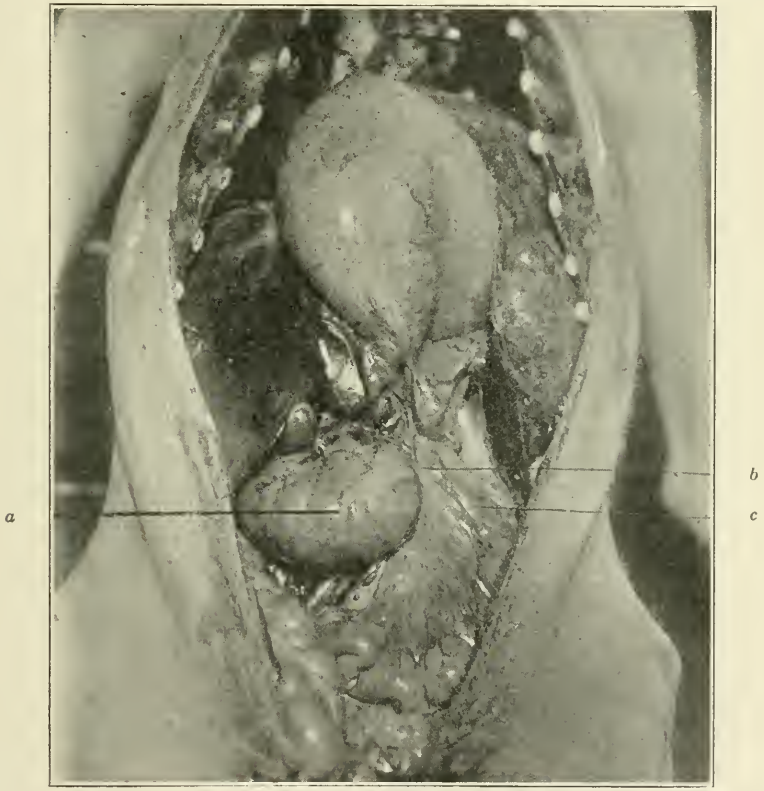

Fig. 2.—View of same case, after the stomach had been turned upward and the transverse colon removed, showing the greatly distended duodenum, the constricting root of the mesentery, and the collapsed first portion of the jejunum. *a.* Dilated duodenum. *b.* Root of the mesentery. *c.* Collapsed jejunum.

color and very emphysematous. Miliary tubercles are numerous beneath the pleura and throughout both lungs. No old tuberculous lesions are found in any part of the lungs. The bronchial and mediastinal lymph nodes are much enlarged and show extensive caseation, but no softening. These caseous nodes extend up along and behind trachea into neck. The spleen is greatly enlarged; it measures 17.5 by 11.5 by 8 cm., and weighs 620 grams. The great size is due to presence of myriads of tubercles, 2 to 4 mm. in diameter. Remaining splenic pulp very hyperemic, so that tubercles stand out sharply. The left kidney weighs 140 grams, the right 185 grams. Few small tubercles in each; capsules not adherent; surface smooth; markings distinct; pelvis and ureters normal. The stomach is enormously enlarged and distended by gas and 650 c.c. of greenish-brown fluid. Greater curvature extends to within 7 cm. of symphysis. Distance from fundus to lowest part of greater curvature is 31 cm. Pylorus in normal position and patulous. Duodenum 7 cm. in diameter and tensely distended with fluid to point where mesentery crosses it. Remainder of small intestine is collapsed and lies chiefly in pelvis. Owing to catarrhal inflammation, slight œdema and absence of gas; the small intestine is heavy and with its mesentery weighs 750 grams. The weight of this mass as it hangs into the pelvis drags heavily on the mesentery and causes firm pressure upon the terminal part of duodenum. A finger introduced into duodenum encounters complete obstruction at this point, which is relieved by raising the mesentery. In this constricting band of mesentery the superior mesenteric artery can be felt as a tense, firm cord. No structural changes in duodenum or pylorus. The stomach wall is very thin. Pancreas normal. The suprarenal bodies are normal except for a single tubercle in the right one. Liver weighs 1600 grams, and is normal except for the presence of vast numbers of tubercles. Lymph nodes about pancreas, lesser omentum, retroperitoneal region, groin, and axillæ are all somewhat enlarged and most of them contain fairly large tubercles. Mesenteric nodes slightly swollen. No tubercles. The other organs are normal.

Microscopically the stomach mucosa shows a fairly marked catarrhal inflammation. The muscular layer seems about half its usual thickness. There is no apparent separation of the muscle bundles, and no structural change appreciable in the fibers.

This case differs from most of those reported (1) in the remarkably short duration of the symptoms, and (2) in the entire absence of vomiting. No immediate cause for the development of the condition could be discovered. The patient had for many days taken only very small quantities of liquid food and that morning had had only a part of a glass of milk. The bowels had moved almost daily up to the time of his death. While there is no certainty that the stomach may not have been dilated before his terminal symptoms began, there had been nothing to suggest this. He had had no gastric symptoms other than anorexia, and, although his abdomen had been examined frequently and carefully, there had been found no increase of stomach tympany, no splashing, no visible peristalsis. During the last week or two of his life he had kept the dorsal position in bed almost constantly. The probable influence of this, and of the almost complete abstention from food, in favoring the production of mesenteric obstruction of the duodenum will be discussed later.

In addition to the case just described I am able, through the courtesy of Dr. James Ewing, to furnish brief notes of another instance of acute dilatation of the stomach.

The patient, a man aged twenty years, was under observation for about six weeks and presented the picture of chronic pulmonary tuberculosis, with hectic fever, chills, emaciation, anorexia, and cough. Aside from occasional vomiting with the paroxysms of coughing, he presented neither symptoms nor physical signs of any stomach disorder. His condition remained practically unchanged until two days before his death, when he suddenly began to vomit large quantities of greenish fluid. His urine became scanty and bloody,vomiting persisted, his condition changed rapidly for the worse, and he died within forty eight hours of the onset of the violent gastric symptoms.

The autopsy revealed, in addition to the lesions of advanced pulmonary tuberculosis, a stomach enormously distended and reaching to within three inches of the symphysis pubis. No lesion was found to explain the enormous dilatation. The small intestine showed no abnormality other than a moderate grade of congestion. There was no peritonitis.

Etiology. The following study of acute dilatation of the stomach is based upon an analysis of 102 cases:

Age. In 90 cases in which the age of the patient is given the percentages according to decades are as follows: First, 4 per cent.; second, 20 per cent.; third, 36 per cent.: fourth, 19 per cent.; fifth, 14 per cent.; sixth, 5 per cent.; seventh, 2 per cent. It will be noted that three-fourths of all the cases developed during adolescence or in early adult life, *i. e.*, between the tenth and the fortieth years. The youngest case was that of a child of three years (Dickinson); the oldest that of a woman of sixty-nine (Kundrat).

Sex. Males were affected in 47 per cent. of the cases; females in 53 per cent.

In studying the state of health of the patients at the time of the onset of the symptoms, and the conditions which seemed to bear on the orgin of the disease, it is seen that the cases fall into several more or less well-defined groups:

1. *Those following Operation with General Anesthesia.* These constitute much the largest group and include 42 cases (41 per

cent.); 15 followed various operations upon the gall-bladder and bile passages (Riedel, Robson and Moynihan, Kehr, and others); 17 developed after other abdominal operations, such as removal of the uterus (v. Herff, Braun), operations on the kidney (Morris, Thomson, Borchardt), etc.; and the remaining 10 cases after operations upon various other parts of the body, chiefly joints. Thus the condition has complicated excision of the knee (Goodhart); of the hip (Jessop); of the elbow (Albrecht); amputation of the thigh (Box and Wallace, Goodhart); amputation of the breast (Albrecht); tenotomy of the tendo Achillis (Müller), etc. It is worthy of note that no instance was found in which acute dilatation had followed operation upon the stomach itself. In almost all the cases in which the nature of the anesthetic used is mentioned it has been chloroform. In one instance, however (Herrick), it is noted that ether (preceded by nitrous oxide gas) was used; and in a number of other cases in which the anesthetic is not mentioned it seems altogether probable that ether was the drug employed.

The time of the advent of the symptoms of dilatation could be determined with reasonable accuracy in 32 cases. In 16 of these they appeared within the first twenty-four hours after operation, and the usual postnarcotic vomiting merged imperceptibly into that of the acute dilatation; in 7 cases the symptoms appeared during the second twenty-four hours, and in 7 others between the third and the seventh day after operation. Once (Robson, Case I) the condition manifested itself, without known cause, on the tenth day of convalescence, and again (Robson, Case III) two weeks after operation. In this latter case the symptoms appeared after eating a raw apple. In a number of the cases the stomach had been seen at the time of operation and nothing abnormal had been noted.

2. *Those Occurring in the Course of or during Convalescence from Severe and Wasting Diseases.* The condition has been met with five times in the course of lobar pneumonia, three times in chronic pulmonary tuberculosis, and once each in general miliary tuberculosis, long-standing hip-joint disease, severe and protracted sciatica, severe chorea, grave anemia, and inoperable sarcoma of the humerus. Meyer has seen it develop in a case of mental disease of long standing (without polyphagia). The symptoms have appeared during convalescence from typhoid fever in four instances. In one of these (Kundrat) they developed immediately upon the patient leaving his bed for the first time. The condition has also been seen during convalescence from severe scarlet fever, from pneumonia, and from some obscure fever. In this group the features common to most of the cases have been long-continued decubitus, prostration, and emaciation.

3. *Cases Developing after Injuries.* Acute dilatation of the stomach has followed injury to the head or spine in four instances.

Rosenheim observed it after a fall upon the shoulder and head (without fracture). In Campbell Thomson's patient it followed fracture of the base of the skull and of the cervical spine. Edmunds records a case of shot wound of the lower dorsal spine, with complete paraplegia, in which thirty-four days after the injury symptoms of acute dilatation suddenly appeared and terminated fatally in nine hours. Erdmann saw the immediate development of dilatation after a fall upon the back in which the body was bent sharply backward. A slight blow upon the abdomen seemed to be the exciting agent in a case of Box and Wallace.

4. *Cases Ascribed to Errors in Diet* (*Dilatatio ex Ingestis*). Indiscretion in diet is given as the direct or contributing cause in fifteen instances; with what justification it is not always easy to determine. Nine times the symptoms developed after a single large meal in persons in good health (Boas, Broadbent, Fränkel, Heine, Hoffmann, Körte, Kirch, Kundrat, and Valsalva). In Broadbent's case the patient had been tramping for two days without food. After a large meal of roast pork he developed severe abdominal pain and vomiting and died at the end of two days. In Heine's case the stomach had been examined a short time before and had been found to be normal in size and in motor function. The symptoms began in Kirch's patient after a supper of two plates of soup and a glass of beer. Six times other possible causes existed along with errors in diet. In Bennett's patient, who was ill of empyema, the symptoms followed the drinking of two bottles of effervescent lemonade. In two instances (Buzzard, Kelling) the patients were encased in plaster jackets (for spinal deformity), and the symptoms appeared after a copious meal. Acute dilatation developed in a woman seven months pregnant after bodily fatigue and the drinking of a glass of cold milk (Dyson). In a case of valvular heart disease dilatation and death supervened after distention of the stomach with food (Goodhart). A patient convalescent from typhoid fever developed evidences of dilatation of the stomach after drinking a very large amount of fluid (Heine).

5. *An Interesting Group of Cases is that Associated with Disease and Deformity of the Spine.* There are 6 such cases; 3 of these (Buzzard,[1] Kausch, 2 cases) were instances of Pott's disease with dorsal kyphosis and (in 2 cases at least) increased lumbar lordosis. In 1 case (Willett) there was a uniform posterior (kyphotic) curve of the whole dorsal and lumbar regions; in another (Kelling) a dorsolumbar scoliosis, and in a third (Kirch) a rachitic dorsal kyphoscoliosis. A further interesting fact is that in 3 of the above cases the gastric symptoms became manifest while the patients were enveloped in a plaster-of-Paris corset (Buzzard, Kelling, Willett).

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[1] This case is often credited to Perry and Shaw.

6. Finally there are 4 cases (W. H. Brown, Miller and Humby, Simon, Wright) in which the symptoms of acute stomach dilatation appeared while the patients were in their usual good health, and for which no cause whatever is assignable. In another case (Schmorl) the symptoms appeared immediately after a violent paroxysm of laughing.

SYMPTOMS. Vomiting, pain, abdominal distention and tenderness, constipation, thirst, scanty urine, and rapid collapse are the chief and common symptoms.

Vomiting is unquestionably the most constant and most important. It has been present in over 90 per cent. of the cases reported. In only 2 cases (v. Herff, Case II; Conner) has its absence throughout been specifically noted, although in 3 other instances (Borchardt, Dickinson, Edmunds) no mention is made of it among the symptoms. The vomiting in most cases appears at the beginning and persists throughout the illness. In a number of instances, however, it has been noted that the vomiting ceased a day or two before death, and once (Bennett) it began only after severe abdominal pain had lasted for five days. "Persistent," "incessant," "uncontrollable," "profuse," are the terms usually used in describing it. The quantity of fluid vomited has varied greatly, but in general it has been noticeably large. Many writers comment upon the surprisingly large amounts brought up. Morris was so struck with the quantity vomited in his case that he looked upon the excessive secretion of fluid as the primary condition and the dilatation of the stomach only as the result, and he proposed for the condition the name of "acute gastrorrhœa." In Miller and Humby's case "five basinfuls" of fluid were vomited during one night, and in a number of the cases as much as several quarts of fluid have been removed from the stomach at one time.

The vomited material is usually of thin, watery consistence. Its color varies much, but is generally some shade of green. Occasionally it is "brown" or "blackish," and in many instances the vomit is described only as "bilious." There can be no doubt that bile is present in the vomit in most of the cases. In several instances small quantities of blood could be detected in the stomach contents. A feature of much diagnostic importance is the fact that although the vomitus has sometimes been foul-smelling, it has only rarely been feculent. In 3 instances only was the vomit noted as distinctly fecal (Kundrat, Lehmann, MacEvitt), and in 1 other (Willitt) it is described as "stinking."

Concerning the chemical character of the fluid vomited or removed by tube but little has been learned. In both of Baümler's cases an absence of free HCl was noted. In one of these lactic acid was present and the fluid showed also the biuret reaction and the presence of a diastatic ferment. Herrick found bile present, a total acidity of 18 units, no free HCl, and no lactic acid. Kelling's case showed no free HCl, but much lactic acid. On the other hand, Heine found a total acidity of 48 units, the presence of free HCl, and an absence of lactic acid. The presence of hydrogen sulphide was noted by Boas and Heine.

Pain. Definite statements as to the presence or absence of abdominal pain are found in only 42 cases. In these pain of greater or less severity was present 35 times. In the remaining 7 cases no pain occurred. The pain was referred usually to the epigastrium or the umbilical region, and in many of the cases was described as very intense.

Tenderness. The presence or absence of tenderness is recorded but 17 times. It was present in 12 and absent in 5 of these cases.

Distention was present in most cases. Its absence was noted only 4 times. It affected chiefly the epigastrium and the region to the left of the umbilicus, but in a few instances it is described as having been almost altogether below the navel. In others it was general and globular. Fagge describes the distention as lying below a line running obliquely downward and to the right to the right iliac region.

In a few instances *muscular rigidity* was noted, but oftener its absence is recorded.

Constipation. In many of the reports no record of the state of the bowels can be found. In 14 cases, however, there was no passage either of gas or feces. In 9, on the other hand, diarrhœa existed. Often the symptoms are described only as being those of intestinal obstruction.

The *temperature,* except in cases in which the symptoms have developed in the course of febrile diseases, has been almost invariably normal or subnormal. In a few of the fatal cases there has been a moderate rise of temperature toward the end.

Thirst is described as intense in many of the cases, as might be expected, and complete suppression of, or great diminution in the amount of, the urine is frequently noted. This is not an invariable rule, however, for in 2 instances with persistent vomiting it is expressly stated that no diminution in the quantity of urine occurred. Indican was present in the urine in some cases.

Persistent hiccough was noticed in a few of the patients, as was also a terminal delirium. Severe muscular cramps throughout the body occurred in 1 case (Wright), but no instance of tetany is recorded.

Splashing sounds and fluctuation waves below the level of the umbilicus aided in the diagnosis in a considerable number of the cases.

Visible Peristaltic Waves. A notable feature of the physical signs has been the absence of visible peristalsis. Only 3 exceptions to this rule could be found. Baümler and Herrick each saw slight peristaltic waves over the stomach. Schultz also saw slight waves on one day, but on the next, when the dilatation had increased, they had disappeared.

Next to the vomiting the rapid development of *collapse* is perhaps the most striking feature of the clinical picture. The sunken eyes, the pinched and livid features, the superficial breathing, and the small, empty, very rapid pulse suggest at once some grave abdominal lesion and serve to explain the common diagnosis of perforative peritonitis or intestinal obstruction.

Duration and Prognosis. Among the 102 cases collected there were 74 deaths (72.5 per cent.) and 28 recoveries. It seems very unlikely, however, that this represents the true ratio between the patients that die and those that recover. There are probably many instances of moderate grade of acute stomach dilatation which get well without being recognized or, at least, without being recorded. In 63 of the fatal cases the duration of the symptoms could be determined with reasonable accuracy, and was as follows: Less than twenty-four hours, 7 cases; one to two days, 11 cases; two to three days, 14 cases; three to five days, 16 cases; five to ten days, 7 cases; ten to sixteen days, 8 cases. In other words, the duration of the symptoms in 75 per cent. of the fatal cases was less than five days. The 2 shortest cases were that of Dickinson (three or four hours) and my own (three hours); the longest that of Kausch (sixteen days). In the patients that recovered the duration of the symptoms varied greatly. In a few postoperative cases these disappeared almost immediately upon change of posture or some other therapeutic measure, but in other instances the dilatation subsided only very gradually (in the course of weeks), and once, at least (v. Herff), a moderate degree of dilatation persisted.

Morbid Anatomy. The statements which follow are based upon an analysis of the records of 69 autopsies.

The most noteworthy feature of the postmortem picture is the enormous size of the stomach. In almost every autopsy protocol the extreme degree of dilatation is commented upon. Often the organ occupies almost the whole of the abdomen. In 1 instance it is described as reaching into the true pelvis. In a number of cases it extended to the symphysis pubis. Once at operation it was mistaken for an immense pancreatic cyst, and as such was evacuated and drained (W. H. Brown). Riedel describes its color as seen at operation as purplish red. In several instances, however, it was grayish or bluish white (Robinson, Fenger, MacEvitt). The distended stomach assumes in most cases a characteristic cylindrical form and is sharply bent into two unequal portions. The larger of these—the cardiac portion—extends almost perpendicularly downward, while the smaller and shorter pyloric limb extends upward and to the right, producing a sharp, angular bend in the lesser curvature and giving to the organ a somewhat V- or U-shape.

Among the whole 69 autopsies definite obstruction at the pylorus was found only three times: once by tumor (Thomson, Case I), once by constriction by a band of mesentery (Riedel, Case III), and once by kinking from adhesions (Riedel, Case II).

The stomach wall is described as abnormally thin in 14 cases. In 3 instances (Albrecht, Case II; Müller, Cases I and II) the wall is mentioned as of normal thickness. In 1 case (Kundrat, Case III) it is described as "thick." In none of the other cases is the condition of the wall mentioned.

A histological examination of the stomach wall is recorded in only a small proportion of the cases. In 1 (Schultz) there were found hemorrhages into the muscular layer, which seemed much thinner than normal and in which the muscle bundles were in many places torn apart and widely separated. In Miller and Humby's case also the muscle fibers were found torn apart. In the few remaining cases, including my own, in which a microscopic examination was made, the musculature presented no appreciable change other than the thinning. The wall in several cases showed many minute hemorrhages in the mucous and submucous layers. In 2 cases (Stieda; Fraenkel, Case II) the mucosa was the seat of small superficial erosions. In 1 instance (Körte) there existed an area of necrosis in the fundus, with perforation of the wall. In a very few cases there were present the lesions of chronic gastritis.

Duodenum. A part or the whole of the duodenum was found dilated in 38 cases (55 per cent.). In almost all of the remaining ones no mention whatever is made of the condition of this part of the intestine, and, as in many of these the autopsy records are very scanty and incomplete, it appears altogether probable that in some of them a dilatation of the duodenum may have been overlooked or, if noticed, not recorded. It seems, therefore, safe to say that in quite two-thirds of the cases, at least, the dilatation is not confined to the stomach, but involves as well a part or the whole of the duodenum.

In 19 of the 38 cases in which the duodenum was dilated the dilatation stopped suddenly near its lower end, at the point where the duodenum passes behind the root of the mesentery, and definite obstruction existed here by the pinching of the gut between the tense mesentery and its contained superior mesenteric artery in front and the aorta and vertebral column behind. That the pinching of the duodenum by the tense mesenteric band is real and serious is abundantly proved by the autopsy findings in Baümler's case, which revealed, in addition to a greatly dilated stomach, a dilatation of the duodenum to the point where it was crossed by the root of the mesentery. At this place the gut was compressed and showed a sharply defined band of bright-red color 2 cm. wide, and corresponding to this ring a necrosis in the mucosa 1.5 cm. wide. Microscopically this band of necrosis was found to be deepest at its centre, where it reached almost to the submucous layer.

In 8 other cases the dilatation ceased at the lower end of the duodenum, but compression by the mesentery was not noticed. These cases are those of Box and Wallace (Cases II and III), Buzzard, Fenger, Meyer, Petit, Schultz, and Thomson (Case V). In Buzzard's and Petit's cases the dilatation was ascribed to a sharp kink at the duodenojejunal junction. In Schultz's case, in which the duodenum had a diameter of 9 cm., the compression at the lower end was thought to result from the weight of the greatly dilated stomach. Meyer suggests the same explanation in his case. It seems very probable from the descriptions that some of these 8 cases may have been instances of mesenteric obstruction.

Three times (Hood, Jessop, Kirch) the dilatation involved not only the duodenum, but the upper few inches of the jejunum as well. In 1 instance (Mahomed) the duodenum and "intestines generally" were dilated. In another case (Kausch, Case I) the first portion (superior limb) of the duodenum was enormously dilated, and at the junction with the descending portion was kinked at an angle of 180 degrees with this latter. In Balster's patient there was an extravasation of blood (of unknown cause) in the wall of the descending limb, which caused constriction of the lumen, with great dilatation above.

The state of the intestines below the duodenum is not noted in many cases; when it is mentioned, however, they are described as empty and collapsed. In only 1 instance (Mahomed) are they spoken of as distended. In almost every case of mesenteric obstruction the small intestine has been found collapsed and lying chiefly in the true pelvis.

The Mechanism of Acute Dilatation of the Stomach. From the time of Brinton this condition has been looked upon by most writers as primarily of paralytic nature. The absence in almost all cases of obvious obstruction at the pylorus, and the fact that a similar paralytic distention of the intestines is a not uncommon phenomenon after abdominal operations and in peritonitis, have served to support this view; as has also the experimental work of Carion and Hallion. In practising, for other purposes, section of the vagi at varying levels, these investigators noted that gastrectasis followed the operation. "After resection of both nerves above the diaphragm we have seen produced in the dog an enormous dilatation of the stomach and of that portion of the œsophagus innervated"—and they add: "If section of the vagus nerves is capable of engendering complete atony and permanent dilatation of the stomach, it is hardly disputable that effects of the same nature could be determined by paralysis or inhibition of the corresponding nervous centres without any previous alteration in the stomach itself."

Accepting the usual teaching that the vagi supply the motor impulses to the stomach musculature, while sympathetic fibers from the dorsal cord, through the solar plexus and splanchnic nerves, carry inhibitory impulses to control such peristaltic movements, the 2 cases (mentioned under Etiology) in which injury to the head has been followed by acute gastric dilatation would seem to be illustrations of such central paralysis of the motor function; and the cases following injury to the dorsal spine might perhaps be looked upon as due to abnormal stimulation of the inhibitory nerves. In the same way the 5 cases which developed in the course of severe pneumonia (Box and Wallace, Herrick, Hood, Lehmann, Thomson) might, it is conceivable, result from the involvement of the vagus trunks in the inflammatory exudate of the posterior mediastinum. It happens that in 4 of the 5 cases there was involvement of both lower lobes, and in the fifth case consolidation of the right lower lobe, with extensive plastic pleurisy over the bases of both lungs—in other words, in all 5 cases the conditions were favorable for the extension of the inflammation to the neighborhood of the vagi. Unfortunately in none of the postmortem examinations of these cases was the question of the involvement of the vagi investigated.

While in a few of the cases it is possible, then, that the explanation of the sudden dilatation may lie in some disturbance of the nerve centres or nerve trunks, this probably does not hold good for most cases. In the great majority of instances the disturbing agent must act directly upon the stomach musculature or its terminal nerve filaments. It has been suggested that the excretion of chloroform by the stomach may explain the large class of postoperative cases (v. Herff). Albu in his case ascribed the paralysis to the absorption of toxins formed by abnormal fermentative processes in the stomach. In other instances blows over the stomach, mechanical injury during operation, local peritonitis, as well as overdistention, severe infections, rapid emaciation, etc., have been regarded as the cause of such paralysis.

The view that acute dilatation depends upon a primary paralysis of the stomach musculature has not been universally held, however. Robson concludes that an analysis of all the cases "would seem to point to a neuroparesis associated with spasm of the pylorus, and Pepper and Stengel also believed some of the cases, at least, to be obstructive, "the pyloric stenosis being due to spasm, the result of acute indigestion or hyperacid secretion."

The theory of pyloric spasm as an explanation for any considerable proportion of the cases seems untenable, in view of the facts that in most of the cases the vomitus contains bile and that in about two-thirds of the cases the dilatation involves also the duodenum.

Morris was disposed to look upon the excessive secretion of fluid ("acute gastrorrhœa") as the primary condition and the dilatation as secondary, but there seems to be little basis for such a view.

With the accumulation of cases of acute gastric dilatation associated with mesenteric constriction of the duodenum, the question has very naturally arisen whether most cases of dilatation might not be due to such obstruction of the duodenum. That many instances of dilatation have not shown this obstruction postmortem would not in itself disprove such a theory, since in the usual manipulation of the intestinal coils the condition might very easily be overlooked unless the possibility of its occurrence were borne in mind.

The whole subject of the mechanism of acute gastric dilatation has been carefully studied by Kelling, and while one may hesitate to accept all his conclusions, his work seems to throw some light upon a problem which at best is complex and puzzling. Kelling starts out with the assumption that, as a prerequisite to the production of acute dilatation, there must be some obstruction to the exit of the stomach contents (fluid and gaseous) *both* upward and downward, for otherwise the overdistention of the organ (by which alone, he thinks, acute dilatation can occur) will be prevented by vomiting and eructation or by the passage of the stomach contents into the intestines. In a series of experiments upon cadavers he found, by inflating the stomach through an opening in its anterior wall, that in a considerable proportion of cases the cardiac orifice was impervious to the passage of air from the stomach into the œsophagus no matter how great the pressure might be; in other cases air would pass only when the intragastric pressure was high, and in still others it passed so freely into the œsophagus that no considerable intragastric pressure could be secured. He found this valve-like closure of the cardia to depend upon certain anatomical relations between the fundus of the stomach and the insertion of the œsophagus. In such cases the cardia can be opened (from below) only by active contraction of bands of muscle fibers running from the œsophagus into the wall of the fundus, and if, by a weakening of these muscles from any cause or by a great increase in intragastric pressure, the active opening force fall below the pressure which maintains the closure of the cardia, any emptying of the stomach through the œsophagus is rendered impossible.

With the œsophagus securely closed by ligature Kelling found, further, by inflating the stomach, that in certain of the cases air passed freely through the duodenum, but that in a majority of instances obstruction occurred by the formation of a sharp kink in some part of the duodenum. Sometimes this kink formed at the junction of the superior with the descending limb, at the point of attachment of the ligamentum heptoduodenale, and above the papilla of Vater; in other cases it occurred below the papilla—usually at the junction of the descending limb with the inferior horizontal portion. The production of such kinks (the possible occurrence of which has long been recognized) seems to depend upon the firmness with which the duodenum is attached to the posterior abdominal wall. In cases in which the gut is relatively loosely fixed it is able to alter its position sufficiently to prevent such kinking.

It is those individuals, Kelling thinks, who have the tendency both to valvular closure of the cardia and to the production of an obstructive kink in the duodenum who are liable, with overdistention of the stomach by gas or food, to the development of acute dilatation.

I have myself repeated Kelling's postmortem experiments in a series of 12 cases (adults dying of affections not involving the stomach or duodenum) with results which differ somewhat from those he obtained. In 7 instances very considerable distention of the stomach occurred before there was any leakage of air through the œsophagus, but such leakage always took place when the air pressure reached a certain height, which, measured by a water manometer, varied between 15 and 26 cm. In the other 5 cases air passed freely into the œsophagus and no distention of the stomach could be produced.

My results in the matter of duodenal obstruction were also somewhat at variance with those of Kelling. With the œsophagus ligated and the stomach inflated, obstructive duodenal kinks were seen in only 3 cases, and in each of these air passed the obstruction when the intragastric pressure measured 22 cm. or less by the water manometer. In each instance the kink formed at the junction of the superior with the descending limb. In 1 of these cases the air, after passing the kink at 20 cm. pressure, found further obstruction at the point of crossing of the root of the mesentery. In 4 other instances obstruction occurred not in the duodenum but at the pylorus, which was tightly closed and required a pressure of from 15 to 25 cm. to overcome it. After passing the pylorus no further obstruction was encountered.

The conditions existing in all such postmortem experiments must of necessity be so entirely different from those which obtain during life, however, as to make it very doubtful indeed if the results so obtained can safely be applied to the explanation of the phenomena of acute dilatation.

More significant, I think, are certain animal experiments made by Kelling and verified by Braun. They found that in dogs upon which a gastrostomy had been done, and which had had time to recover from the effects of the operation, it was impossible to produce a condition of acute dilatation by inflating the stomach, for whenever a certain degree of distention was reached a vomiting reflex was excited and the stomach was emptied by vomiting and eructation. When, however, the animal was narcotized the stomach could be distended to the point of bursting without the least escape of air through the œsophagus and without any reflex attempt at regurgitation or vomiting.

That the development of acute dilatation of the stomach may be favored by anatomical peculiarities which lead to obstruction both at the cardia and in the duodenum seems altogether likely, but that such double obstruction is *necessary* to its production (as Kelling believes) seems to be clearly negatived by the fact that in many instances of typical and extreme dilatation vomiting and eructation have been present throughout the whole course of the disease.

While there is good reason then to doubt that obstruction at the cardia, or failure of the vomiting reflex, is essential to the development of acute dilatation, there is much evidence to show that obstruction below the stomach is present in very many of the cases and that it plays a role of considerable importance in the production of such dilatation.

Among the 69 autopsy reports studied an associated dilatation of the duodenum was noted 38 times (55 per cent.), and of these 38 cases 25 showed some form of obstruction below this dilatation (see Morbid Anatomy). In some of the remaining 13 cases the records are very incomplete and unsatisfactory, and it is impossible to say whether obstruction existed or not. But even if none of these cases is included, and if no allowance is made for the fact that occlusion by kink or by pressure might very easily be overlooked at the postmortem table, there still remains the fact that in 36 per cent. of all the autopsies definite obstruction was found in some part of the duodenum.

Further evidence of the frequency and importance of obstruction below the stomach is to be found, it seems to me, in the fact that the quantity of fluid vomited or removed from the stomach has been, in most cases, so enormous, and that this excessive amount of fluid can be most satisfactorily explained (as Müller has shown) by the assumption of some obstruction high up in the intestines by which the usual reabsorption of the (normally) large amount of fluid secreted by the stomach, pancreas, and liver is prevented. An analysis of the 25 cases in which some form of duodenal occlusion was shown to exist gives the following results: In 19 instances the dilatation ended at the point where the duodenum is crossed by the root of the mesentery, and the obstruction was ascribed to constriction by the tense mesentery. In 2 other cases (Meyer, Schultz) the dilatation extended to about the same point, but the obstruction was believed to be due to pressure of the displaced pyloric portion of the stomach. Twice (Buzzard, Petit) the occlusion was caused by a sharp kink at the duodenojejunal junction; once by a kink at the junction of the superior with the descending limb of the duodenum (Kausch), and once by an extravasation of blood (of unknown cause) between the mucous and muscular layers of the wall of the descending portion of the duodenum (Balster).

Mesenteric Obstruction of the Duodenum. The frequency with which mesenteric compression of the duodenum has been found associated with acute dilatation of the stomach and duodenum demands that this remarkable condition shall now be discussed in some detail. I have collected 18 fatal cases from the literature (Albrecht (2), Baümler, Binswanger, Kausch, Kelynack, Kundrat (3), Lehmann, Müller (2), Philippe, Robinson, Schmorl (Kelling), Schnitzler, Stieda, Walzberg).[2]

In addition to these fatal cases there are, among the cases of acute gastric dilatation which recovered, several (Bäumler, Müller, Robson, Schnitzler, Walzberg) in which the clinical picture was that of mesenteric obstruction of the duodenum, and in which a cure was effected by treatment (chiefly postural) directed toward the relief of the supposed mesenteric compression.

In view of the presence of bile and a diastatic ferment in the stomach contents and of the result of postural treatment in Baümler's second case it can hardly be doubted that the diagnosis of mesenteric obstruction was correct. The other cases referred to are scarcely less convincing.

In looking over the cases of mesenteric obstruction cited it will be seen that the condition, in its etiology, symptoms, duration, and prognosis, does not differ from that of acute gastric dilatation in general. Even the characteristic symptom of bilious vomiting has been present in most cases of acute dilatation of the stomach in which no mesenteric compression was noted.

Among the 69 fatal cases examined mesenteric obstruction was thought to exist 19 times (27.5 per cent.). In 19 other cases, as has been said, dilatation of the duodenum was found at autopsy, and, although in some of these instances other causes for the dilatation were discovered, in a number of them it seems very probable that mesenteric compression may have been present. If to these are added the several cases with recovery in which there is good reason to believe that the same condition existed, it becomes evident that obstruction of the duodenum by the mesentery must be regarded as a factor in the development of a large proportion (perhaps from one-third to one-half) of all the cases of acute gastrectasis.

The subject of the production of this form of duodenal incarceration has been carefully studied by Albrecht, who points out that the terminal portion of the duodenum, where it passes behind the root of the mesentery and lies upon the vertebral column and aorta, has under normal conditions not a round but a distinctly flattened circumference, owing to the pressure of the overlying mesentery and its contained superior mesenteric artery. This normal constriction can readily be seen in a cadaver in the dorsal position, and if a finger is passed into the duodenum and at the same time gentle traction downward is made upon the mesentery, the constriction becomes more distinct and is very evident to the examining finger. If now traction upon the mesentery is made in the direction of the axis of the pelvis—that is, downward and backward—the constriction may be converted into complete occlusion. With the small intestine lying altogether or chiefly in the pelvis, traction would be made upon the mesentery in just the direction needed to produce such occlusion. The presence of the small intestine in the true pelvis seems then to be a *sine qua non* to the production of such mesenteric occlusion of the duodenum, and this view is borne out by the autopsy findings of the recorded cases. In order that the intestine may make its way into the pelvis and there exert traction upon its mesentery, it is necessary that it contain little or no gas and that the mesentery be of suitable length—long enough to allow the intestine to hang in the pelvis without being supported by the pelvic floor.

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[2] A brief synopsis of these cases will be found in the Trans. Assoc. Amer. Physic., 1906, xxi, 601.

It is possible to demonstrate on the cadaver, as Albrecht and others have done, that traction on the mesentery in the direction of the axis of the pelvis may produce obstruction in the duodenum which will be impervious to water under considerable pressure. Box and Wallace, however, conclude from their experiments that the great distention of the stomach and the obstruction in the duodenum that can be produced in cadavers, in the dorsal position, by water pressure, depend not upon constriction by the root of the mesentery, but rather upon the direct pressure of the heavy stomach upon the duodenum where it crosses the lumbar vertebræ.

This question I have attempted to study in a series of 10 adult subjects. With the bodies in the dorsal position and the organs in place, the small intestine was removed and the mesentery gathered up into a mass and fastened to a cord which was passed into the pelvis and out through the anus and was allowed to hang free. To this different weights could be attached. The stomach and duodenum were then filled through the œsophagus, by a small tube of water from the tap, and the pressure necessary to overcome the duodenal obstruction estimated by connecting the tube with a mercury manometer.

The results differed greatly in the different cases. In 3 instances the obstruction seemed to be due almost entirely to the weight of the distended stomach upon the end of the duodenum, and with this raised very little interference with the flow of water into the jejunum was produced by a weight of 500 grams or even 1 kilogram attached to the mesentery. In each of these 3 cases a certain degree of gastroptosis existed. In the other cases a load of 500 grams (the approximate weight of the empty small intestines) produced an obstruction, to overcome which required a water pressure varying from 10 to 48 mm. of mercury. In some of these cases the stomach gradually distended to a point where it lay upon the duodenum, and by its weight increased somewhat the obstruction caused by the tense mesentery. In other instances the dilated stomach did not rest upon the duodenum and exerted no influence whatever upon the mesenteric obstruction.

The conclusion seems justified that in a certain proportion of normal individuals a pull upon the mesentery approximating, in direction and force, that which might be exerted by the empty small intestines hanging in the true pelvis can produce obstruction at the lower end of the duodenum which will require very considerable force to overcome.

The conditions essential to or which favor mesenteric obstruction may for convenience be divided into two groups: those which aid in the descent of the intestine into the pelvis, and those which favor the more complete occlusion of the duodenum.

For the entrance of the intestine into the pelvis three things, at least, seem necessary: dorsal decubitus, an intestine nearly or quite empty of gas and feces, and a mesentery of suitable length—long enough to permit the intestines to slip into the pelvis, and yet not so long that they may be supported by the pelvic floor.

It is possible that the frequency of such obstruction after operation (in 8 of the 19 cases) may depend in part upon the fact that at such times the intestines have been kept empty by fasting, purges, enemas, etc.

Other conditions which seem to favor the descent of the gut into the pelvis are chiefly those which disturb the normal equilibrium of the intra-abdominal pressure. Kundrat believed that a lax, flabby abdominal wall acted so by failing to hold the intestines in their usual position. Lehmann, in his case, which developed in the course of a double lower-lobe pneumonia, suggests that the weight of the hepatized lungs and the violent coughing efforts may have aided in forcing the small intestine into the pelvis. In the remarkable case of Schmorl the symptoms followed immediately upon a violent paroxysm of laughing. In one of Kundrat's cases they suddenly appeared in a typhoid-fever convalescent who had left his bed for the first time.

The duodenum is rendered more liable to serious compression, Schnitzler believes, by any unusual prominence (lordosis) of the lumbar vertebræ, or by an abnormally low position of the last portion of the duodenum, since the vertebræ become increasingly prominent as they descend.

Increased lordosis existed in 2 of the recorded cases (Kausch, Robinson), as well as in several other instances of acute gastric dilatation in which no mesenteric obstruction was recognized.

The same result was brought about in Philippe's case by a mass of clotted blood which lay behind the terminal portion of the duodenum and pushed it forward. He also demonstrated experimentally that by the injection of a quantity of tallow behind the lower part of the duodenum complete arteriomesenteric obstruction could be produced even with the small intestine not in the pelvis.

The line at which the third portion of the duodenum crosses the lumbar vertebræ varies much. Fawcett and Blachford, in studying this point recently, found the position of the lower border of the duodenum to vary between the upper border of the second lumbar vertebra and the disk between the fifth lumbar and the sacrum. Most commonly it crossed the third lumbar vertebra. In general the point of crossing was lower in females than in males. In view of the fact that the anterior curve of the lumbar vertebræ reaches its maximum at the fourth vertebra, it is obvious that a duodenum which lay upon the fourth vertebra would be more liable to serious compression by the mesentery than would one which crossed, say, the second vertebra. There has been some differences of opinion as to whether the constriction of the duodenum is caused by pressure of the whole root of the mesentery or chiefly only by the superior mesenteric artery which runs in it. The frequent use of the term "arteriomesenteric obstruction" indicates that the artery has often been looked upon as playing an important role in the production of the obstruction, and there can be no doubt that to the examining finger in the duodenum, with traction upon the mesentery, the most resistant portion of the constricting band is the tense, cord-like strand which represents the artery; but, on the other hand, in Baümler's fatal case the band of hyperemia and necrosis in the duodenum corresponded in width and position to the whole root of the mesentery and not to the artery alone.

From a consideration of the conditions just enumerated it is not difficult to see how a transient occlusion of the duodenum might occasionally occur; but why such an occlusion should become a permanent, unyielding obstruction, with its train of violent symptoms and usually fatal outcome, is, as Albrecht has pointed out, by no means easy to explain. It seems very unlikely that such a pinching of the duodenum should always result in complete and permanent incarceration. It is more natural to suppose that it sometimes produces only a temporary occlusion which passes unrecognized. What is it then that occasionally renders such an obstruction permanent and insurmountable?

The almost constant presence, in the fatal cases, of an enormously dilated stomach has led to the very general belief that this is the factor of chief importance in the production of a permanent incarceration. By the rapid increase in the size of this organ the intestine would tend to be crowded more and more into the pelvis, and, once there, would be prevented from escaping. At the same time the weight of the heavy stomach upon the mesentery would be likely to render futile every effort on the part of the duodenal and gastric musculature to overcome the obstruction.

While there is general agreement that the greatly enlarged stomach is of much importance in rendering the incarceration more complete and permanent, there is still a sharp difference of opinion as to whether the dilatation of the stomach or the obstruction of the duodenum be the primary act in the vicious circle by which the dilated stomach increases the duodenal obstruction, and this latter in turn augments the dilatation of the duodenum and the stomach.

Von Herff, Stieda, and Neck believe the dilatation of the stomach to be always primary and the mesenteric constriction to be purely a secondary happening. On the other hand, Müller and Lehmann regard the duodenal obstruction as of prime importance and the first act in the chain of events.

That this latter view is correct, for some cases at least, seems altogether probable from the fact that in 2 of the cases of mesenteric obstruction (Kundrat, Case II; Philippe) no mention whatever is made in the autopsy reports of any dilatation of the stomach. Also in such cases as that of Schmorl, in which the symptoms followed directly upon a violent paroxysm of laughing, it seems probable that the trouble began with the sudden constriction of the duodenum by the mesentery.

On the other hand, it is easy to see how a considerable dilatation of the stomach, either acute or chronic, might favor the production of mesenteric obstruction, and, when this has occurred, prevent its spontaneous recovery. Albrecht believes that in many of the instances of apparently acute gastrectasis there has been a pre-existing chronic dilatation which has converted a transient mesenteric constriction of the duodenum into a permanent and complete obstruction, and that this, in turn, has brought about a sudden increase in the gastric dilatation with its train of alarming symptoms. Unfortunately for this theory there is little evidence in either the clinical or the autopsy records to support the idea that chronic gastric dilatation is a frequent antecedent of mesenteric obstruction.

Chronic Mesenteric Obstruction of the Duodenum. Kundrat and Glénard held that a persistent, incomplete obstruction of the duodenum by the root of the mesentery was not uncommon and led to a gradual dilatation of the duodenum and stomach. Albrecht cites 2 cases which he thinks may have been instances of this condition, and Robinson asserts that in the course of several hundred postmortem examinations he has met with fifteen or twenty examples of gastroduodenal dilatation due to such incomplete obstruction.

Nothing is known of the clinical manifestations of such chronic mesenteric obstruction, and, in view of its possible importance, it is very desirable that the attention of surgeons be directed to the need of careful examination of the lower part of the duodenum and the mesenteric root in operations affecting that part of the abdomen. It is quite possible that some of the instances of duodenal dilatation, to the frequency of which operators in this country

(Finney, Mayo, Ochsner) have recently called attention, may be due to such chronic mesenteric obstruction.

DIAGNOSIS. It is interesting, in looking over the records of the reported cases of acute gastric dilatation, to note that whereas the first case seen by most of the writers has passed unrecognized, subsequent cases observed by the same men have almost always been correctly diagnosticated during life. In other words, the condition is usually not difficult of recognition if the possibility of its occurrence be but held in mind. Intestinal obstruction and perforative peritonitis have been the conditions with which it has most frequently been confused. Once at operation the hugely dilated stomach has been mistaken for and treated as a pancreatic cyst. In some of the postoperative cases the persistent vomiting has been looked upon as only an exaggerated form of the usual postnarcotic vomiting.

The sudden onset of severe and continued vomiting, and especially the vomiting of very large amounts of bilious, non-fecal, fluid, should always suggest the possibility of acute gastric dilatation. A normal or subnormal temperature, rapid emaciation and collapse, obstinate constipation, and abdominal pain, tenderness and distention are other very common symptoms; but it must be emphasized that any one, or most, of these may be lacking in a given case. In two of the recorded cases vomiting has been absent throughout. In a few instances there has been neither pain, tenderness, nor distention. Occasionally diarrhœa has replaced constipation. The diagnosis is made definitely by the demonstration of the greatly dilated stomach by means of careful physical examination and especially by the use of the stomach tube. The existence of mesenteric obstruction of the duodenum is made probable by the demonstration, in the contents of the dilated stomach, of bile and of pancreatic juice, by the absence of distinctly fecal vomiting, and by the satisfactory results of postural treatment. It is possible that an examination of the pelvis through the rectum or vagina might show there the mass of collapsed intestine. Kelling suggests, as a means of determining whether or not complete obstruction exists at the lower end of the duodenum, the use of a high injection of a large quantity of concentrated (6 per cent.) salt solution. The usual effect of this is to empty the small intestine as well as the colon, and the presence or absence of fresh bile in the returned fluid would determine the question as to whether or not the duodenum was pervious.

TREATMENT. The effectiveness of treatment in acute dilatation of the stomach will depend, in large measure, upon the time at which it is begun. When the stomach has become dilated to its maximum and the patient has reached a state of extreme prostration and collapse, treatment has often been unsatisfactory and unavailing. On the other hand, when the disease has been recognized before this terminal stage was reached the results of treatment, in most cases, have been very gratifying.

The first step in treatment, whatever the cause of the dilatation may be, is the prompt and complete emptying of the stomach by means of the stomach tube. Owing to the often enormous size of the organ and the flaccid state of its walls, this is not always a simple task. The tube must be made to reach the most distant and dependent parts of the stomach, and changes in the patient's posture may be necessary in order that all the contents may be reached. This emptying of the stomach should be repeated at regular intervals, usually several times in the twenty-four hours. Food or drink by mouth should be absolutely interdicted. The excessive loss of fluid should be met by large saline enemas or by hypodermoclysis. Nutrition should be maintained as well as possible by rectal feeding.

In a considerable number of the cases the above-mentioned treatment has been all that was needed to effect relief of the symptoms and gradual recovery, but in view of the fact that some form of duodenal obstruction has been shown to exist in 36 per cent. of the fatal cases, and has probably been present in many others, the likelihood of such obstruction has to be considered in every case. If such obstruction is due only to a duodenal kink or to the weight of the dilated and overloaded stomach, the mere emptying of the stomach and the keeping of it empty may suffice to relieve the obstruction. It has been shown, however, that three-fourths of all the cases of duodenal obstruction have been caused by constriction by the root of the mesentery, and for the relief of such obstruction something more than the emptying of the stomach is needed. Schnitzler was the first to treat successfully a case of acute gastric dilatation on the assumption that the symptoms were due to mesenteric obstruction. The patient was changed from the dorsal to the belly position and all the symptoms promptly ceased. After several hours he was able to resume the dorsal position. The abdominal distention had disappeared, vomiting did not return, feces and flatus passed freely, and the patient made a good recovery.

In Baümler's case (already cited) the patient was kept in the knee-elbow position for a quarter of an hour in each two hours, the rest of the time lying on his belly. The symptoms disappeared promptly and the patient recovered.

The experience of Müller (Case IV) was even more striking. In this patient vomiting persisted in spite of lavage and elevation of the pelvis. It ceased immediately, however, when the belly position was taken. After two hours a dorsal posture was tried, but vomiting began again at once. The patient lay on her belly through the night and slept well. Next day she was able to take and retain food in this position, but every attempt to lie on her back was followed by vomiting. On the following day, however, she could lie on her back comfortably and thereafter had no further trouble.

Walzberg records a very similar experience (Case II). Lavage having been of no avail, the patient was turned upon his right side and three hours later upon his left side. Vomiting ceased immediately. Two days later, on attempting to lie upon his back, nausea returned and the lateral position had to be resumed. The patient made a good recovery. In one of Robson's patients (Case IV) the symptoms were relieved by keeping her in the belly position.

These results are so striking that is seems but fair to give every case of acute dilatation of the stomach the benefit of the doubt, and to try the effect of such postural treatment as seems most likely to displace the intestine from a position in the pelvis and to relieve a possible mesenteric constriction of the duodenum.

It should be noted, however, that in 1 case (Borchardt) the symptoms seemed to be made worse by the use of the belly position.

The relief of acute gastric dilatation by operation has been attempted a number of times. In 7 instances (W. H. Brown, Box and Wallace, Appel, Hoffmann, Jessop, Kirch, Wright) the stomach was opened and evacuated, but none of the patients recovered. Gastroenterostomy has been performed twice (Kehr, Körte), and both times unsuccessfully. Petit found at operation a sharp kink at the duodenojejunal junction. This he relieved by lifting up the jejunum and stitching it to the transverse mesocolon. The symptoms disappeared and the patient recovered. The advisability of a gastroenterostomy is open to serious question. There is quite general agreement that it should be employed, if at all, only as a last resort. In most cases it is unnecessary, and in those patients who have resisted all other methods of treatment it is doubtful, Borchardt thinks, if any good can be accomplished by operative measures.

SUMMARY. Acute dilatation of the stomach is a disease of considerable clinical importance and is deserving of wider recognition. It develops under a great variety of conditions, but is especially frequent after operations with general anesthesia. In a majority of the cases there has been an associated dilatation of the duodenum.

The mechanism of acute gastric dilatation is not yet clearly understood, but in a large proportion of cases (perhaps one-half) the dilatation is associated with, and seems to result from, some form of obstruction in the duodenum.

Much the commonest cause of such duodenal obstruction is the constriction of the lower end of the duodenum between the root of the mesentery, which crosses in front of it, and the vertebral column. Such constriction can occur only when the mesenteric root, with the superior mesenteric artery which runs in it, is stretched tightly across the duodenum; and this is possible, apparently, only when traction is exerted by the small intestine hanging over the brim of the pelvis. The development of mesenteric constriction is favored, therefore, by whatever facilitates the entrance of the small intestine into the true pelvis (dorsal decubitus, a mesentery of suitable length, an empty and collapsed state of the gut, etc.), and whatever renders the duodenum more liable to compression (increased lordosis of the lumbar vertebræ, low position of the duodenum, etc.). Great dilatation of the stomach would doubtless increase the liability to mesenteric obstruction by crowding the intestines into the pelvis, and would tend, by preventing the escape of the gut from the pelvis, to render such obstruction, when once formed, more complete and permanent.

Whether the dilatation of the stomach or the mesenteric obstruction of the duodenum constitutes, usually, the first step in the vicious circle by which both conditions are made worse is still uncertain, but there is little doubt that in some cases, at least, the duodenal obstruction is primary.

The symptoms and physical signs of acute gastric dilatation are usually so distinct that the diagnosis should not be difficult if the possibility of such occurrence be but remembered.

In spite of the high mortality (72 per cent.) among the reported cases, there is much evidence to show that the disease can usually be controlled and cured if the diagnosis be made and suitable treatment begun before complete collapse of the patient has occurred.

The following list of 82 cases,[3] in addition to the 2 cases reported herewith and the 18 cases of mesenteric obstruction already cited, constitutes the material upon which the present study is based:

Albu, Appel, Balster, Baümler, Bennett, Bettmann, Boas, Borchardt, Box and Wallace (3 cases), Braun (5 cases), Broadbent, H. H. Brown, W. H. Brown, Buzzard (Perry and Shaw), Dickinson, Dyson, Edmunds, Erdmann, Fagge (2 cases), Fenger, Fisher, Fränkel (2 cases), Goodhart (4 cases), Heine (2 cases), v. Herff (3 cases), Herrick (2 cases), Hoffmann, Hood, Hunter, Jessop, Kausch, Kehr (2 cases), Kelling, Kirch, Körte, Lieutaud, MacEvitt, Mahomed, Meyer, Miller and Humby, Morris (2 cases), Müller (2 cases), Petit, Riedel (3 cases), Robson and Moynihan (4 cases), Rosenheim, Schnitzler, Schultz, Simon, Thomson (5 cases), Valsalva, Walzberg, Willett, Wright (2 cases).

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THE OPERATIVE TREATMENT OF FRACTURES, ESPECIALLY OF THE LONG BONES.

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When a surgeon is called upon nowadays to treat a patient with a fractured femur, for example, the problem which immediately confronts him is whether to operate or not to operate. There was a time long gone by when the question of operation in such cases never arose unless it was a question of amputation or resection; operation for the purpose of putting the broken fragments in proper relationship with one another was not considered, since the proper adjustment of the fragments was thought to be done by extension, counterextension, and manipulation. The Röntgen rays have shown that most fractures of long bones set in this way have resulted in shortening due to overlapping of the fragments.

The question may be asked, Why should we operate on fractures which are almost certain to get well without operation? The answer is, For several reasons: (1) To prevent deformity of a limb, especially shortening, which is of the greatest importance in the lower limbs; (2) to prevent loss or impairment of function, as of pronation or supination; (3) to obviate delayed union or ununited fracture; (4) to give opportunity to repair the soft tissues—nerves, muscles, and bloodvessels; and (5) because, as a rule, it is the only way to get a perfect result. It is now generally agreed that for a fractured patella or olecranon the operative is the best treatment.

I believe that perfect adjustment in fractures of the long bones is seldom accomplished without operation, except in green-stick fractures and some transverse fractures in which the fragments may be only partially displaced and have not overridden, as in some Colles fractures. It is also often impossible to ascertain, after a supposed reduction, whether or not apposition has been obtained. In the case of a broken femur surrounded by muscles from two to four inches in thickness, who by palpation can tell anything about the relations of the fragments? We resort to measurement: a mistake of one-half to three-fourths of an inch may easily be made on account of the broad or flat bony prominences between which we measure. The grating of bony fragments and the result of measurement may indicate complete reduction, but its stability cannot be depended on: the fragments may be touching each other over the smallest fraction of an inch, and this contact may be broken at any moment by the slightest movement of the limb, and shortening may be reproduced. By exposing the site of the fracture by incision, and then having extension and counterextension made while I manipulated the naked ends of the fragments, I have repeatedly demonstrated the great difficulty of reducing by extension and counterextension fractures of the humerus, radius, ulna, femur, and tibia in which the ends of the bones overlapped. In transverse fractures it has often been necessary to bring the ends out of the wound, in partial contact with one another, making an angle like the rafters of a roof, then by straightening the limb the ends of the fragments would be brought in full contact with each other. In oblique fractures, even with the aid of the lion-jawed and other bone-holding forceps, I have sometimes been unable to get perfect reduction and have had to fasten the fragments together with a fourth of an inch shortening. In a patient with fracture of the middle of the shaft of the femur treated for a few days on the double inclined plane, operation revealed an overriding of two inches and a separation of the fragments from each other by muscular tissue one inch in thickness.

It is the opinion of many that Buck's and other kinds of extension can correct the shortening. It may diminish the shortening, but in the majority of cases I believe it cannot correct it fully. I treated a man, sixty years old, with a fracture in the upper third of the shaft of the femur, on a double inclined plane, with extension in a line with the femur with a weight of sixty pounds, without getting complete reduction. The method by operation was explained to him, but he preferred the other and made a good recovery, with one inch and a half shortening of the femur.

If there is anything in the theory that union of bone is hindered or prevented by the intervention of soft tissues, too much motion, or lack of accurate approximation, operation properly performed surely must often prevent delayed union. I say properly performed, because otherwise it seems possible to increase the danger of delayed union. For example, too extensive detachment of the soft parts with the periosteum from the fragments may cause necrosis. In some cases in which union has taken place in bad position and it has been necessary to operate, delayed union may result. The resources of the tissues seem to have been exhausted in the formation of the callus for the first union; whence they are unequal to the second task imposed upon them.

The dangers of the operative method must be carefully considered. That means the dangers which come by reason of the operation, and not those which attend fractures treated in other ways: (1) Anesthesia. This is not peculiar to the method by operation, but is in general use in the treatment of fractures. (2) Fat embolism. As shown by Connell and others, this occurs in nearly all fractures of large bones containing much fat, and is probably dangerous only when the amount of fat taken up by the capillaries is large—a condition likely to be brought about by much manipulation of the fragments, and therefore equally as likely to result from efforts to reduce a fracture by the blind as by the open method. (3) Extensive detachment of the periosteum and other soft parts must be avoided as already mentioned. (4) Infection. This is the chief danger, as the others mentioned are common to any method in which anesthesia is used, and in modern surgery this should not be great.

Technique. Of the various means used to hold the fragments of bones together, such as plates, screws, nails, ivory pegs, bone ferrules, animal sutures, and wire, I believe the wire the best in a large majority of cases. It is less bulky, it can be applied with much less injury to the soft parts than some of the others—the metal plates, for example—and its action in holding the bones together continues longer than any of the other methods except the bone ferrule. The nails and screws soon cause softening of the bone in which they are embedded, and the bone ferrule is not satisfactory according to my limited experience. It is not always necessary to wire the fragments or to use sutures. Transverse fractures, once perfectly reduced, remain in position by the use of splints, but it is safer to wire them. Kangaroo tendon, silk, and heavy catgut may do in some cases, but as a rule wire is better, because it is stronger and is not absorbed too soon. No matter how much care may be used to keep the ends of the bones together, some force, as the contraction of muscles, will tend to displace them, and this force may continue to act after animal sutures have been absorbed. Recently I have been using iron wire. It has the advantages over silver wire of being much stronger and cheaper, it does not seem to irritate the tissues or to be more often followed by suppuration than silver wire, and I believe it is possible in time for the iron wire to corrode away and disappear.

Wire may be used in several ways: (1) In transverse fractures each fragment is drilled through at right angles and two strands of wire are used (Fig. 13). (2) Only one hole is drilled in each fragment and only one loop of wire is used (Fig. 6). (3) The drill-holes do not pass through the bone, but only enter the medullary cavity (Fig. 5). The first of these methods gives the greatest security against displacement. (4) In oblique fractures the fragments may be drilled through while in position, the wire passed through, and then carried several times around the fragments. (5) The wire may simply be carried several times around the fragments after adjustment, without drilling (Figs. 2, 3, and 4), or it may be carried around the fragments in position at two or more points (Fig. 2).

For carrying wire around bones so as to injure the soft parts as little as possible, I have had a strong, full-curved needle made in three sizes. For holding the fragments in position while drilling or wiring, Foraboeff's bone-holding and the lion-jawed forceps are indispensable. For drilling the bone a drill with a handle like a gimlet is the most satisfactory. All hemorrhage having been arrested and the wound closed, the best splint is plaster-of-Paris, but a window must be cut—through which one may observe the progress of the wound in the soft parts.

Cases. In my note-books I find histories of 102 cases of fracture in which I have operated, as follows: Superior maxilla 4, inferior maxilla 12, clavicle 5, humerus 10, radius and ulna together 5, radius alone 1, ulna alone 10, femur 13, patella 20, ruptured patella tendon 1, ruptured quadriceps extensor 2 (these three cases are placed next to fractures of the patella, to which they are nearly allied), tibia 16, and fibula 3. There were two deaths, both after operation on the neck of the femur; they will be considered later.

Superior Maxilla. The fractures of the superior maxilla were bilateral and complicated with fracture of the bodies of the palate bones and of the pterygoid processes of the sphenoid, the line of fracture passing through the superior maxillæ from the front backward through the nasal fossæ and the antra of Highmore, above the palate processes, through the body of the palate bones and the pterygoid processes, so that the entire alveolar arch and palate dropped slightly and were freely movable. In one case the two maxillæ and palate bones were separated from their fellows in the median line. The causes of these fractures were street railway accidents in 2, and blows across the front of the upper jaw in 2. There was no difficulty in obtaining union, though one was complicated with an abscess of the antrum of Highmore.

Lower Jaw. The fractures of the lower jaw were all through the body of the bone and the alveolar process, most of them oblique; the transverse fractures were usually treated with bandages and splints only. The line of fractures should be exposed by an incision along the lower border of the body of the jaw and the soft parts stripped off until the bone is exposed on each side of the line of fracture sufficiently for drilling a hole. Care should be taken not to injure the mental nerve at its exit from its foramen, nor the branches of the cervicofacial division of the seventh nerve near the angle of the jaw. It may be necessary to ligate and divide the facial artery. The holes in the fragments should be drilled as near to the line of fracture as is safe, and low enough not to injure the teeth roots or the inferior dental nerve. The wire should then be introduced, twisted until the fragments of bone are in apposition, the ends cut off, and the twisted part turned beneath and to the inner side of the bone. The teeth should be wired, if practicable, passing the wire around the teeth two teeth distant from the line of fracture, twisting the wire on the outer side of the teeth, and turning the end down against the gum so that it will not irritate the lip (Fig. 1). These twelve patients all made good recoveries.

Clavicle. The fractures of the clavicle, five in number, were all treated by incision, exposing the parts, putting the fragments in place, and holding them by wrapping them with catgut, kangaroo tendon, or silver wire. This was done by means of a large, full-curved needle, carrying the thread around from three to eight to ten times. Care was taken to separate the muscle attachment as little as possible, both to avoid necrosis and also to afford tissue which would prevent the thread which is passed through it from slipping. In no case was it necessary to drill the fragments. The results were all good, and there was no shortening (Figs. 2, 3, and 4).

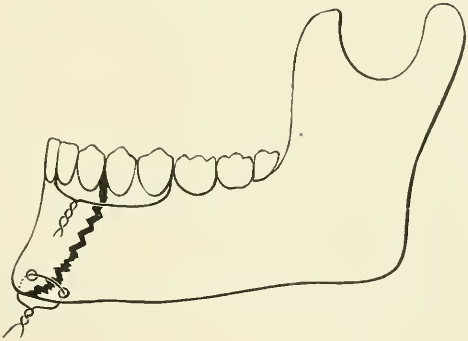

Fig. 1.—Fracture of left side of the lower jaw, showing the wires in place—one through holes drilled in the lower part of the body of the jaw, the other around the teeth.

Humerus. Of these, 10 were operated on: 2 of the neck, 1 of the middle third of the shaft, 3 supracondylar, 2 T-fractures, and 2 condylar fractures. One of the neck fractures (Case VIII) occurred in a man, twenty-five years old, who fell from a step-ladder a height of about four feet, and struck on his right shoulder. Examination showed swelling and the projection upward and inward, in front of the acromion process, of the upper end of the lower fragment, as in epiphyseal separation; a diagnosis of fracture of the anatomical neck was made. The parts were exposed by an incision from the anterior part of the acromion process downward in line with the humerus, splitting the fibers of the deltoid to the outer side of the long head of the biceps and the attachment of the pectoralis major, cutting and tying a few branches of the circumflex arteries, and exposing the circumflex nerve, but pushing it out of the way. On retracting the soft parts the fracture was exposed. The principal line of fracture ran from the anatomical neck in front downward, inward, and backward, for an inch and a half obliquely, through the bone, separating the tuberosities from each other—the greater tuberosity remaining with the upper fragment, the lesser forming the upper end of the lower fragment. A fragment about half an inch square was broken from the greater tuberosity, but was held in position by the fibrous tissue. The joint was not opened. The displacement was easily reduced by traction on the arm; a hole was drilled in each fragment into the medullary cavity, and a piece of silver wire was put in and twisted, thus holding the fragments in exact apposition (Figs. 5 and 6). The wound was closed without drainage and the arm was bound to the side with a sling for the forearm. There was primary union of the soft parts, and forty-five days later there seemed to be bony union with good result—motion, shape, etc., being normal. A skiagram showed the parts in normal position.

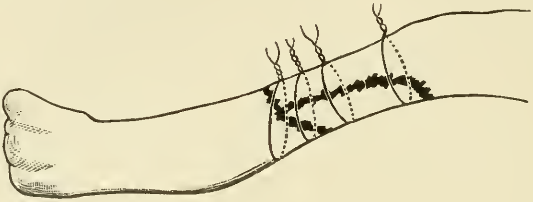

Fig. 2.—Fracture of the left clavicle at the junction of the middle and the outer third, with the production of three fragments. These were adjusted and held in place by four kangaroo ligatures encircling the fragments; two of the ligatures were placed obliquely. (Case 1. book 2, page 49.)

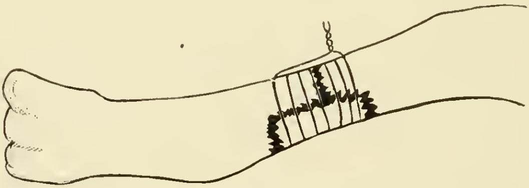

Fig. 3.—Fracture of the left clavicle (three pieces), showing the silver wire carried around the fragments. (Case IV, book 6, page 130.)

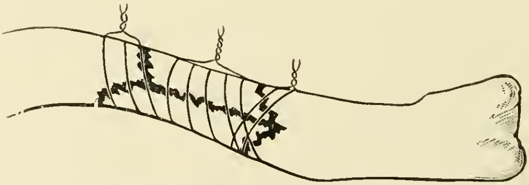

Fig. 4.—Fracture of the right clavicle (the middle third broken into four fragments), showing wire at the ends passed obliquely around the fragments and through the muscular attachments to prevent slipping. (Case V, book 6, page 136.)

Case IX was a compound transverse fracture of the surgical neck of the left humerus in a boy eighteen years old. The wound in the soft parts was two inches below the shoulder-joint and a little to the outer side of the anterior surface. The wound was enlarged above and below, about in line with the middle of the acromion process along the outer surface of the humerus, splitting the deltoid, securing the branches of the circumflex arteries, and avoiding the nerve. There was overlapping of more than an inch—the lower fragment lying to the outer side of the upper fragment. The line of fracture was transverse, with a short spicule attached to the inner side of the lower fragment. There was considerable difficulty in getting the ends together in spite of traction and the use of bone-holding forceps. Once put together there seemed to be no tendency to displacement, but a hole was drilled in each fragment from side to side entirely through the bone, and heavy silver wire was passed through and twisted on the outer side. The wound was closed without drainage and dressed. There was primary union of the soft parts, and at the end of forty-five days bony union seemed to be good, motion, length, shape, etc., being normal (Fig. 7).

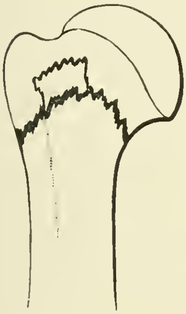

FIG. 5

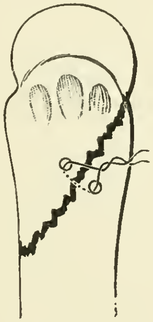

FIG. 6

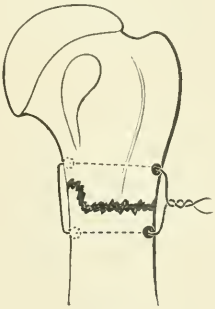

FIG. 7

FIG. 5.—Oblique fracture of the neck of the right humerus, anterior view. (Case VIII, book 6, page 101.)

FIG. 6.—Oblique fracture of the neck of the right humerus, external view, showing the length and direction of the line of fracture, and the wire in place. (Same as Fig. 5.)

FIG. 7.—Left humerus, anterior view, showing fracture of the surgical neck with the wire in place. (Case IX, book 6, page 121.)

A fracture of the middle third of the shaft was the only one which gave a bad result. This was in a sailor, forty-eight years old, who received the fracture two and one-half years before he came to me. It was operated on soon after it was received, before I saw him, but no union resulted. During the year he was under my observation, two operations were performed. The first consisted in freshening the ends of the bone and fastening them together with iron-wire nails. A small amount of bone formed, but no union took place; so that a second operation was done, this time using a bone ferrule. This remained in nearly five months and then was removed without having caused any union, although a slight amount of bone had formed. The patient declined further operation and when last seen had a false joint, a skiagram of which is shown in my book.[1]

The supracondylar fractures were treated by incision through the triceps muscle on the back of the arm, or between the brachialis anticus and supinator longus in front, and wiring of the fragments with or without drilling. All gave good results. The T-fractures and condylar fractures were exposed by one of the incisions already given and the fragments fixed by wire or nail. One case is given as an example:

Case II. Book 3, page 9. A boy, seventeen years old, fell and struck on his right elbow, causing a T-fracture of the humerus. Incision was made between the supinator longus, and the biceps and brachialis anticus, being careful not to injure the musculospiral nerve. The lower end of the upper fragment was displaced forward and inward and the condyles were split apart. The fragments were adjusted and the condyles held in place by a steel drill driven through the skin, the external condyle, and deep enough into the internal condyle to hold fast. The external condyle and the lower end of the upper fragment were then wired together after drilling a hole in each (Fig. 8). The drill was removed at the end of seventeen days. Good union followed and almost normal motion—flexion being normal, but extension only 135 degrees. There was considerable formation of callus.

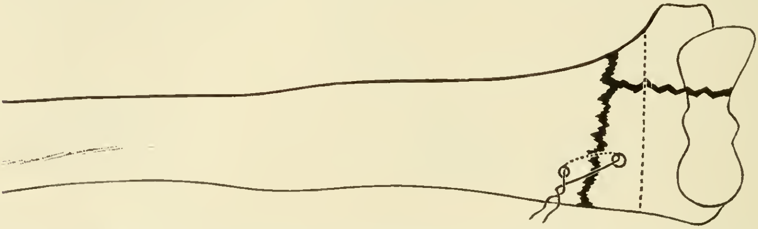

Fig. 8.—Right humerus, anterior view, showing drill through the external condyle into the internal condyle and the external condyle united to the shaft. The dotted line represents the drill. (Case II, book 3, page 9.)

Radius and Ulna. In treating fractures of the radius and the ulna, it is of the first importance to preserve the function of rotation—pronation and supination—by accurately adjusting the fragments. In fractures of the radius just above the insertion of the pronator radii teres, the upper fragment is likely to be rotated outward (supinated) and the lower fragment rotated inward (pronated). In all, 16 operations were done—5 on the radius and ulna together,

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[1] Manual of Surgery, p. 253.

1 for Colles' fracture, 1 for fracture of the shaft of the ulna, and 9 for fracture of the olecranon.

In operating on these bones the greatest difficulty is encountered in connection with the radius; the ulna can be exposed, without trouble and without endangering any important structures, by an incision at any point along its posterior border, but the radius is covered by thick muscles in its upper half and by important tendons and the radial nerve in its lower half. Two incisions are necessary when operating on both bones—one for each bone. In fracture of the upper third or of the head of the radius, the incision is best made over the outer and posterior part of the forearm from the external condyle downward over the head of the radius and in the axis of its shaft, dividing the skin and fascia, finding the intermuscular space between the extensor communis digitorum and the extensor carpi ulnaris, and dividing or separating the fibers of the supinator brevis until the bone is sufficiently exposed. The posterior interosseous nerve should be avoided. Branches of the posterior interosseous recurrent artery and other vessels from the anastomosis around the elbow-joint require ligation.

To expose the radius in the middle third, one should make an incision behind the outer border of the forearm along the inner side of the extensor carpi radialis longior, enter the space between the extensor carpi radialis brevior and the extensor communis digitorum until the attachment of the extensor ossis metacarpi pollicis to the radius is exposed. In order to expose the bone sufficiently it may be necessary to separate the attachments of this muscle and those of the pronator radii teres, and, in front, of the flexor longus pollicis. For fractures in the lower third, including Colles', the radius may be exposed by an incision along the outer border of the radius from the styloid process upward, taking care not to injure the radial nerve, which is superficial. The crossing of the carpal extensor tendons by those of the thumb makes it somewhat difficult to expose the radius in the lower third. The best plan is to separate the tendons where it seems easiest and safest in order to expose the fracture. In Colles' fracture, I found it easier to open the "snuffbox"—the space between the extensor longus and the extensor brevis pollicis tendons—exposing the tendons of the extensor carpi radialis longior and brevior, which were retracted to the inner or outer side as might be most convenient.

Olecranon. All fractures of the olecranon do not require operation. Cases are seen in which there is no separation of the fragments, although there is a false point of motion readily detected by making lateral pressure. The fibrous expansion of the triceps tendon, corresponding to the quadriceps expansion about the patella, remains unruptured and holds the upper fragment in place. Separation of a quarter of an inch or more requires operation.

The site of fracture should be exposed by a longitudinal incision over the olecranon. The elbow-joint is usually opened and is often filled with blood clots, which should be washed away with salt solution. The fragments should be adjusted and can usually be fixed in position by sutures of kangaroo tendon through the fibrous tissue covering the bone. Wire may be used, especially when the soft parts are destroyed to such an extent that sutures will not hold. The wire should not enter the joint, the fragments being drilled obliquely as in the patella, two holes in each fragment; or if preferred, the fragments may be drilled laterally where they are thicker and a single strand of wire used. I have used about equally kangaroo tendon and wire, with good results in all. The limb should be dressed and the forearm carried in a sling at right angles; it is not necessary to put up the forearm in extension.

Femur. Thirteen fractures of the femur were operated on: 4 of the neck, 6 of the shaft, and 3 of the lower extremity, including 2 T-shaped fractures and 1 epiphyseal separation. It may be well to give a brief account of these cases as showing both the good and bad results following treatment:

Case I.—A man, aged twenty-five years, sustained a fracture of both femurs in the middle third, the left being compound. The left femur (the compound fracture) united in the usual time, but with two inches of shortening, while the right femur refused to unite. It was operated on, a false joint being found. One inch of bone was sawed from each fragment in order to make the femur correspond in length with its fellow; the bones were drilled and fastened with one strand of silver wire. The patient made a good recovery, with legs of equal length, but his stature had been diminished two inches.

Case II.—A man, aged twenty-five years, sustained a fracture of the right femur in the middle third—upper part. He was operated on the same day through an external incision. The fracture was almost transverse; each fragment was drilled laterally and united by one piece of heavy silver wire. Suppuration followed, but final recovery, with good result, was secured.

Case III.—A girl, aged eleven years, had an epiphyseal separation of the lower end of the left femur, the result of a wagon-wheel passing over the limb. The usual symptoms presented: great swelling of the knee, floating and prominence of the patella, with an abnormal depression above it, hardness in the popliteal space, and shortening of the femur (Fig. 9). Attempts to reduce the displacement without incision failed, so the parts were exposed by an incision along the outer side of the knee. The patella was found resting on the epiphysis and the epiphysis on the anterior surface of the lower end of the diaphysis. Reduction was accomplished with some difficulty by flexing the leg, making traction on the flexed leg, and by pressure. The wound healed without trouble, after which the patient passed from under my observation.

Case IV.—A woman, aged sixty-five years, fell, causing a T-shaped fracture of the lower end of the left femur. Attempts to reduce the fragments failed, so that an incision was made along the outer side of the thigh and knee in front of the biceps tendon, exposing the lines of fracture. The fracture was just above the condyles and transverse except for a long spicule which projected from the upper fragment and was embedded in the soft tissues just above the patella. The fractured surface of the condyles presented backward toward the popliteal space. The condyles were split apart, but were held together by the soft tissues. A hole was drilled through the upper fragment and one through each condyle anteroposteriorly; a piece of silver wire was passed through the holes in the condyles and through the hole in the upper fragment, using a piece of wire for each condyle, but both pieces passed through the one hole in the upper fragment (Fig. 10). Plaster-of-Paris was applied with the knee slightly flexed. There was no trouble, and the result was good.

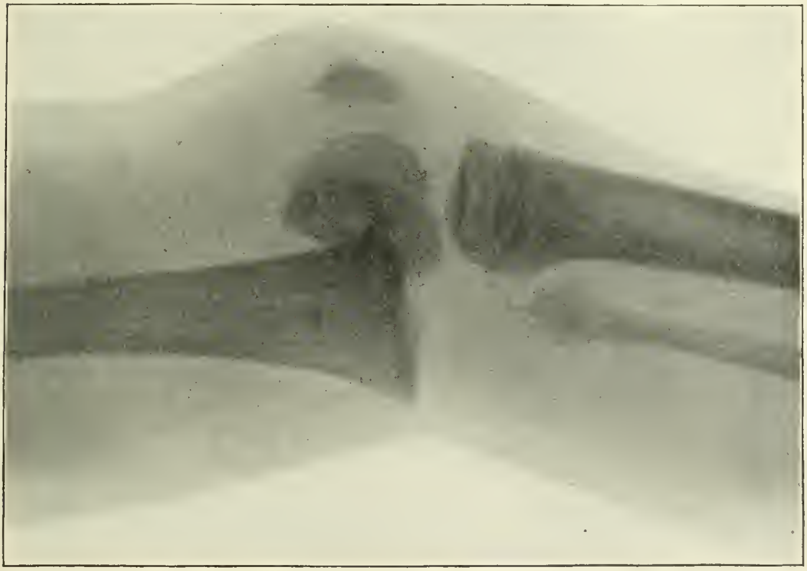

Fig. 9.—Epiphyseal separation of the lower end of the femur.

Case V.—A man, aged twenty-eight years, fell from a platform and suffered a compound T-shaped fracture of the right femur—the lower end of the upper fragment projecting through the skin of the front and inner side of the thigh about two inches above the knee. The incision was enlarged above and below, whereupon the fracture was found to be oblique from above downward and forward. There was also a line of fracture between the condyles, which were movable one on the other, but did not fall apart laterally.

The fragments were accurately adjusted and wrapped with silver wire (Figs. 11 and 12). A good result followed.

Case VI.—A girl, aged six years, sustained a fracture of the left thigh in the upper part of the middle third; she was treated by vertical suspension; after six weeks there was union with two inches of shortening. Through an external incision a fracture, oblique from above downward and outward, was found. The union was broken up, the parts almost perfectly reduced, and wrapped with silver wire. Healing and union followed, but there was half an inch shortening.

Case VII.—A woman, aged sixty-three years, had fractured the neck of the left femur seven years before and had been treated for some years for sciatica and other troubles before the diagnosis of fracture was made. Operation was undertaken on account of great and constant pain. An anterior incision exposing the hip-joint was made; no union; no callus was found; the head and neck were softened and were removed in fragments. The result was good—complete relief from pain and improved locomotion.

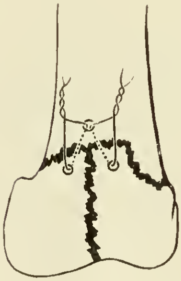

Fig. 10

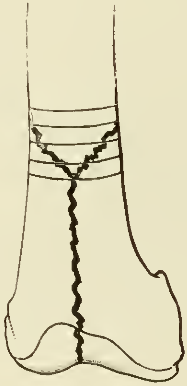

Fig. 11

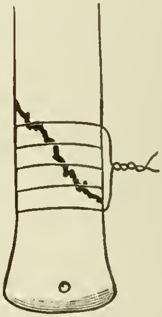

Fig. 12

Fig. 10.—Anterior view of T-shaped fracture of the left femur, showing wires in place. The projecting spicula was cut off. (Case IV, book 4, page 12.)

Fig. 11.—Anterior view of T-shaped fracture of the right femur, showing the wire in place. (Case V, book 5, page 94.)

Fig. 12.—Lateral view of the fracture shown in Fig. 11.

Case VIII.—A man, aged twenty-four years, sustained a compound fracture of the right femur in the middle third, a fragment projecting through the skin of the outer and posterior surface of the thigh. The wound was enlarged and a second incision to the outer side of the rectus muscle was made. There was considerable laceration of the muscles and three loose fragments of bone were removed. The lower fragment was posterior and external to the upper fragment and separated from it by muscular tissue at least one inch in thickness. The fracture was transverse. The ends were drilled and wired together end-to-end. The patient had a severe attack of suppurative tonsillitis four weeks after the operation. The wounds in the soft parts healed without trouble and there was considerable callus formed by the end of the second month, but there has been delay in ossification, so that even now, eight months after the operation, there is slight motion at the point of fracture, which very slowly diminishes.[2] The femur is full length.

Case IX.—A man, aged twenty-one years, fractured his right femur in the upper part of the middle third. An external incision exposed a very oblique fracture over two inches long from above downward and inward, the lower fragment lying in front and to the outer side of the upper. The fragments were adjusted and wrapped with wire. Union followed without trouble in five weeks, but there was half an inch of shortening, evidently caused by slipping of the fragments. It would have been better to drill the fragments, pass the wire through, and then wrap well with the wire. This would probably have prevented shortening.

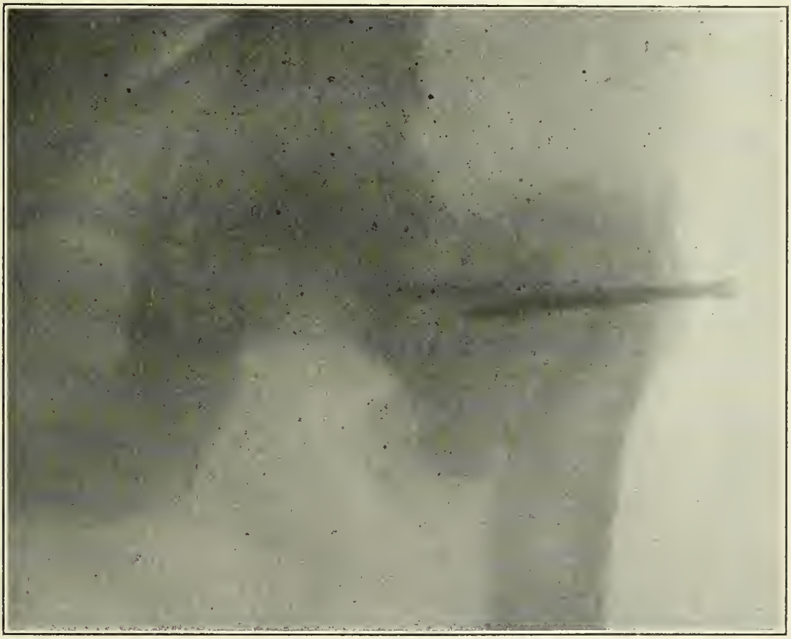

Fig. 13.—Skiagram of a fracture of the base of the neck of the femur, showing the iron nail used to retain the fragments in place.

Case X.—A man, aged thirty-one years, fell from a height of ten to twelve feet and struck on the right trochanter major, causing a fracture of the base of the neck. An anterior incision exposed the neck and upper part of the shaft, showing the line of fracture to correspond with the anterior intertrochanteric line, outside the capsule of the joint and leaving the trochanter major attached to and forming the summit of the lower fragment. The lower fragment had slipped up about one inch, but by traction this was brought down. An incision was made through the skin over the trochanter and a three-inch iron-wire nail was driven through the trochanter at its lowest part, into the neck of the femur (Fig. 13). There seemed to be good union at the end of four weeks, but the patient was not permitted to walk until six weeks had elapsed, and was told not to bear any weight on that leg until three months had passed. The result was good; about one-quarter inch shortening; motion and function of the joint are normal. The nail is still in the bone.

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[2] This patient had fully recovered a month later.

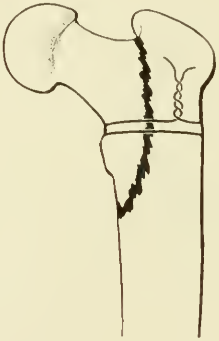

Fig. 14.—Anterior view of an intertrochanteric fracture of the left femur, showing the fragments adjusted and two turns of heavy wire in place. (Case XI, book 6, page 104.)

Case XI.—A woman, aged seventy-five years, suddenly sat down on the floor and fractured the left femur. Under chloroform the upper end of the femur was exposed by an anterior incision. There was fatty degeneration of the muscles and the bone was thinner and more porous than normal. The line of fracture extended from the inner side of the trochanter major downward and inward, a little to the outer side of and diverging from the anterior intertrochanteric line, reaching the inner side of the shaft nearly an inch below the trochanter minor—thus separating the trochanters. The lower fragment lay to the outer side of and behind the upper fragment. The capsule was not opened. No difficulty was met with in adjusting the fragments accurately and in carrying a piece of heavy silver wire twice around the fragments between the trochanters (Fig. 14). The wound was closed without drainage. That night the patient became delirious, the pulse rapid and weak, and death occurred forty hours after the operation. Death was thought to be due to fat embolism lodging in the brain. Unfortunately, no necropsy was made.

Case XII.—A boy, aged eleven years, had his left femur fractured in the lower part of the middle third. An incision along the outer border of the rectus muscle exposed the fragments. There was overlapping of two inches, the lower fragment lying to the inner side and posterior to the upper. The line of fracture was nearly transverse. The ends of the fragments were adjusted with considerable difficulty, then two holes were drilled through each fragment—one from before backward, the other from side to side—and two pieces of wire were put in and twisted. The wound was closed without drainage. A good result was obtained in fifty days, with no shortening or deformity (Fig. 15).

Case XIII.—A man, aged about fifty years, broke the neck of his right femur. An anterior incision showed the line of fracture almost exactly along the anterior intertrochanteric line, the head, neck, and lesser trochanter being separated from the shaft and greater trochanter. The fragments were adjusted and drilled, the holes going only to the interior of the bone, and wired. The wound was closed without drainage. Three days later the patient had a general convulsion. Next day the wound was opened and considerable thin fluid or serum with offensive odor escaped. The patient did not improve; his mental condition was confused, the pulse rapid, and death occurred five days after the operation and forty-eight hours after the convulsion—probably from fat embolism in the brain. No necropsy was permitted.

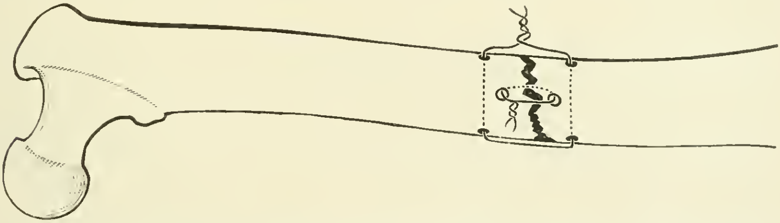

Fig. 15.—Anterior view of a fracture of the middle third of the left femur, showing the fragments adjusted with two strands of wire through holes at right angles to each other. (Case XII, book 6, page 108.)

Both these deaths have been attributed to fat embolism, as the symptoms were similar and in general indicative of that condition, although in neither case were there pulmonary symptoms. Case XI might have been attributed to chloroform poisoning, but ether was used in Case XIII. Every surgeon knows that death is not infrequent as a result of fractured femur, probably caused most frequently by fat embolism, and it is quite likely that the ordinary efforts to reduce the fractures in these two patients without operation would have been followed by the same unfortunate result.

For exposing the femur, I prefer the following methods:

Upper Third. Make an incision, four to eight to ten inches long, from half an inch below the anterior superior spinous process of the ilium downward, in line with the outer border of the patella, cutting into the angle formed by the divergence from each other of the sartorius and tensor vaginæ femoris, to the outer side of the rectus, between it and the gluteus medius and minimus. If the fracture is below the trochanters, the vastus externus must be drawn outward and the fibers of the crureus divided and retracted to expose the bone. From three to six branches of the external circumflex artery require ligation. To examine the neck of the femur within the capsule, it is necessary to separate the psoas and iliacus from the capsule and draw them inward with the rectus, and then split the capsule in line with the long axis of the neck.

Middle Third. Make an incision along the middle third in the line which separates the anterior from the external surface of the thigh, find the outer border of the rectus muscle, draw it inward and the vastus externus outward, and divide the fibers of the crureus, which are closely attached to the bone, until it is exposed to a sufficient extent.

Lower Third. Make an incision from the anterior portion of the outer condyle of the femur upward along the outer side of the thigh, exposing and splitting the vastus externus until the bone is exposed. Several arteries from the anastomosis around the knee will require ligation. Care must be taken not to injure the popliteal vessels and nerves or inadvertently to open the synovial cavity of the knee.

Patella. Twenty operations were done for fracture of the patella; the results were all good. One patient only had fibrous union, and this was the result of a Barker operation—a subcutaneous surrounding of the fragments by a silk ligature passing through the joint. The usual operation was a horseshoe incision, wiring the fragments after drilling, union of lateral fibrous expansion with kangaroo tendon, and closure without drainage. The patients were confined to bed for two or three weeks; care in using the knee was insisted upon, motion being limited for three months by means of a bandage or pad.

In addition, there were two ruptures of the quadriceps extensor and one of the patellar tendon; in all three cases the tendon gave way at its attachment to the bone. Kangaroo tendon was used to suture the part in position. The results were good.

Tibia and Fibula. Sixteen tibias were operated on and three fibulas, with good results; but one is still under treatment. Many of these were compound comminuted fractures. Usually, only the tibia was wired when both bones were broken and the ends of the fibula simply adjusted, sometimes wrapped with wire (two cases), but never drilled. To expose the bones an incision is made half an inch to the outer side of the crest of the tibia. Often both bones can be treated through this incision. Care must be taken not to injure the anterior or the posterior tibial vessels and nerves. If a separate incision is necessary to expose the fibula, this may be made at any point along the line of the fibula from the head to the external malleolus, entering the space between the extensor longus digitorum on the inner side and the peroneal muscles on the outer side, being careful not to injure the musculocutaneous nerve, or, if the operation is near the head of the fibula, the peroneal nerve. Often the position of the incision is determined by a wound already existing in the soft parts. In one case the styloid process and insertion of the biceps were broken from the fibula by muscular action (wrestling). Suture with kangaroo tendon gave a good result.

LEUKEMIC BLOOD PICTURE IN A CASE OF FRACTURE OF THE ANKLE.

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Reports upon the blood findings in cases of fracture are so scarce that it is difficult to form a definite opinion of the frequency with which abnormalities occur. Cabot[1] states that a study of thirty-eight cases, by himself, Hubbard, and Blake, of which fifteen were complicated, showed nothing abnormal. From the figures given, however, it is clear that in a certain percentage of the cases the leukocytes were increased. Ewing and Da Costa give no original observations, and Grawitz also makes no special mention of the findings in fractures. A survey of the journal literature of the past few years likewise brought no data to light. Hastings writes me that in two cases he has seen "myelemia," with myelocytes between 3 and 7 per cent., the one being a severe crush of the thigh (and femur), and the other one of the forearm, with no break in the skin and no evidence of infection.

In view of these meager data the following case report may not be out of place: The patient was a colored man, aged forty-five years, who was admitted, on October 18, 1905, to Dr. Johnson's service at the Maryland General Hospital, with a compound fracture of the right ankle. Careful questioning did not bring out any information which would suggest that the patient had been the subject of myeloid leukemia previous to the accident. He had noticed no lump in the abdomen and had been in his usual strength up to that time.

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[1] Pathology of the Blood, 5th ed. p. 400.

Differential counts were made on October 31, November 2, 4, and 6, and showed an increase of the polynuclear neutrophiles (79 to 83 per cent.) and low eosinophile values, but no myelocytes or other unusual condition.

Cellulitis developed, and on November 15 the leg was amputated in the lower third. The day before the amputation the leukocytes were much increased (50,000 estimated) and a differential count revealed a state of affairs which was typical of myeloid leukemia: small lymphocytes, 3.0 per cent.; large mononuclears, 2.5 per cent.; polynuclear neutrophiles, 56.2 per cent.; eosinophiles, 6.2 per cent.; mast cells, 17.5 per cent.; neutrophilic myelocytes, 15.0 per cent.; eosinophilic myelocytes, 1.2 per cent. While counting, 400 leukocytes, 22 normoblasts and several megaloblasts were seen.

These findings, which were first reported by the senior students who had charge of the hematological observations in the case, were verified by my assistant, Dr. Langley, and myself. The spleen, which had not been examined before, was now readily palpable at the costal border.

Unfortunately no blood specimens were taken again until November 21. At that time the leukemic blood picture was much less pronounced. The leukocytes had diminished considerably in number, and myelocytes, while still present, were less numerous. The mast cells had fallen to 4 per cent., and only an occasional normoblast was seen. After that the myelocytes gradually disappeared, and when the patient was discharged the blood picture was entirely normal.

An examination on April 23, 1906, showed 30 per cent. of small lymphocytes, 10 per cent. of large mononuclears, 59 per cent. of polynuclear neutrophiles, 4 per cent. of eosinophiles, and less than 1 per cent. of mast cells. There were no myelocytes and no nucleated red cells.

An examination of the bone-marrow of the amputated foot was unfortunately neglected.

To recapitulate we have a blood picture, occurring in a patient after a compound fracture of the leg, at a time when cellulitis had developed, which bears every mark that is typical of the myeloid type of leukemia. After a week it wanes, and with the recovery of the patient there is a return to normal conditions. The degree of splenic enlargement, when I first saw the patient, a few days after the discovery of the remarkable blood findings, was not at all proportionate to the blood picture, as we generally see it in myeloid leukemia, but not inconsistent with that diagnosis. At the time I was inclined to think that the case was a true leukemia and that the accident had possibly occurred during a "normal" interval. But the subsequent course of the myelemia inclined me to abandon this view. The return of the mast cells to the normal especially speaks against this possibility. Ehrlich long ago pointed out that the increase of the mast cells is the most constant and characteristic symptom of myeloid leukemia, and I have pointed out at another place that this increase will persist even though the spleen may return to normal size and the blood picture clear up otherwise. Nevertheless, we must assume a *vis a tergo*, and notwithstanding the well-known effect of septic infection upon the character of the leukocytosis under ordinary circumstances, I believe that this factor must be viewed directly or indirectly as a causal element after all. It is known that in osteomyelitis, myelocytes may appear in the circulating blood in large numbers, although this does not occur in every case. An increase of the mast cells and the simultaneous occurrence of nucleated red cells, on the other hand, have not been observed. I can imagine, however, that the findings may differ with a varying localization of the inflammatory process, and that extensive involvement of red marrow may give rise to a different picture than would result if the fatty marrow were largely affected. This idea would, in a manner, presuppose the origin of the marrow elements from the affected structures, which would, on the face of it, seem unlikely. On the other hand, one might imagine that the extensive destruction of red marrow in the injured bones, especially after development of the infection, might lead to a stimulation of the red bone-marrow at large and a resultant functional hyperactivity and the mechanical discharge of bone-marrow elements into the circulation. In this sense the temporary myelemia in my case could be regarded as the leukocytic counterpart to the "crises" affecting the red cells, which are occasionally observed in certain types of severe anemia. This view is the one to which I incline. The case is certainly unique and is reported with the idea of bringing out records of similar cases, which have not yet been reported in the literature.

THE LUYS URINE SEPARATOR.

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The history of separators for the intravesical separation of the urine from the two kidneys dates from 1890, when Lambotte,[1] of Brussels, first introduced an instrument which proved unsuccessful. In 1897 Neumann[2] was also unsuccessful in producing a separator for use in the female. Harris,[3] of Chicago, next introduced his segregator, which was untrustworthy, and caused much pain to the patient. In 1901 Luys,[4] of Paris, introduced his separator. The large caliber of the early instrument limited its use to the female. Later the caliber was made smaller, so that it could be employed in the male and the female. In 1902 Cathelin,[5] of Paris, introduced still another segregator, but this seems to be more complicated than the Luys, and causes more pain.

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[1] Etude sur la taille du rein. Jour. de méd. de chir. et de phar., Bruxelles, Septembre, Octobre, et Novembre, 1890.

[2] Ein einfache Methode den Urine beider Nieren bei Weibe gesondert aufzufangen. Deutsch. med. Woch., October 21, 1897.

The largest number of cases published is 210—by Luys himself.[6] In none of these cases was the cystoscopic examination methodically used. In addition to these, important work has been done by Zuckerkandl, of Vienna, and his assistant, Lichtenstern;[7] the late Mikulicz,[8] of Breslau; Israel,[9] of Berlin; Emile Burckhardt,[10] of Bâle, and Bickerstith,[11] of Liverpool. There are a number of other articles, mainly by French, German, and English writers, but none that I can find from America.

Next to Luys, Lichtenstern (Zuckerkandl's assistant) quotes the largest number of cases, twenty-three in all, in which the separator was employed. This series is of especial interest to me, since it was in Zuckerkandl's clinic that I first saw the separator used, and learned the technique of its use.

In 14 of these 23 cases the findings of the separator were confirmed by operation, either before or after the urinary separation. In the remaining 9 cases the findings so corresponded with the symptoms and physical signs as to admit of no reasonable doubt of their accuracy. Lichtenstern says: "The separation is so exact that not only may the possibility of organic admixture be completely eliminated, but also chemically we may find a marked difference on both sides, as, for example, in pyonephrosis we often observe a difference in the reaction of the urine of the two sides."

Bickerstith quotes 9 cases, in all of which the separator findings were confirmed by operation. Luter, assistant to Burkhardt, quotes 7 cases, in 4 of which the results were confirmed by operation or autopsy.

Caspar,[12] of Berlin, says: "Segregators can occasionally be used with benefit, and in default of the possibility of ureteral catheterization are to be used and recommended. But they are not as exact for all cases as catheterization is, and on this account are inferior. The sovereign means of diagnosis remains with ureteral catheterization; only when this is not practicable may a segregator, either Luys or Cathelin's, be tried." Kummell,[13] of Hamburg, says, "No separator or segregator can give absolutely accurate results." Von Frisch,[14] of Vienna, Posner,[15] and others have given like opinions.

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[3] The Use of the Urine Segregator in the Diseases of the Urinary Tract, N. Y. Med. Rec., April, 1898.

[4] La séparation de l'urine des deux reines, Assoc. Fran. d'Uro., Paris, Octobre, 1901.

[5] Le diviseur vesical gradué à sondes metalliques lateralis mobils, Assoc. Fran. d'Uro., Paris, Octobre, 1902.

[6] La séparation de l'urine des deux reines, Paris, 1904.

[7] Erfahrungen über Harnsegregation. Wiener klin. Woch., 1904, No. 29.

[8] Manuscript letter to Dr. Luys from Gottstein, assistant to the Surgical Clinic, Breslau.

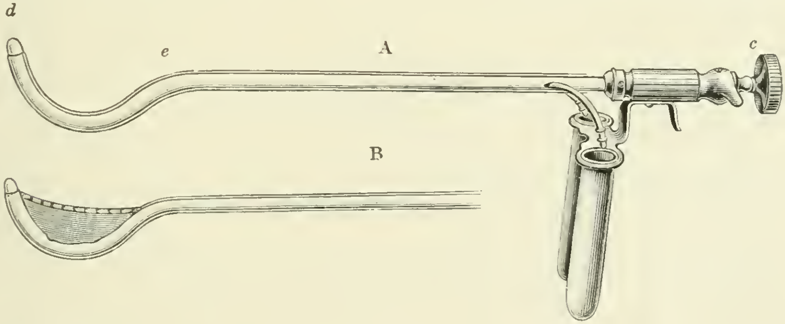
[9] Manuscript letter to Dr. Luys from Dr. Frank, of Berlin, in the service of Prof. Israel, Berlin.

[10] Ueber den Harnscheider von Luys. In Correspondenz-Blatt f. Schweizer Aerzte, 1904, No. 18.

[11] Intravesical Separation of the Urines, Lancet, March 28, 1904, and Intravesical Separation of the Urines, Brit. Med. Jour., October, 1904.

[12] Handbuch der Cystoskopie, Leipzig, 1905.

The Instrument. The Luys instrument (see figure) when ready for introduction into the bladder (A) has the form of a Béniqué metal sound and corresponds to 21, French scale. The portion from *d* to *e* (A) is the intravesical part. The entire instrument may be separated into three portions, one central and two external. A long slender glove-finger of rubber is fitted over the central portion, which in its intravesical part contains a fine chain. By a mechanism at *c* (A) the chain may be tightened and the rubber covering it put on a stretch, so forming a diaphragm which, when the instrument is in position, divides the bladder into halves (B). The lower portion of the rubber diaphragm comes in contact with the bladder wall at *f* (B). The two external portions of the separator are tubes through which the separated urine flows. These tubes have each three fenestrations on the side toward the rubber diaphragm for the entrance of the urine from the ureters.



When the separator has been introduced and the diaphragm adjusted, the empty bladder by virtue of its muscular action grasps the separator, the right ureter emptying its urine on one side close to the diaphragm, and the left on the other side.

Technique of Separation. The instrument must be examined before and after using.

Before Using. (1) To see that both tubes of the separator are permeable; that their lumen is not at all narrowed by dirt, rust, etc.

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[13] Münch. med. Woch., October 17, 1905, p. 2057. Report from Kongress der Internationalen Gesellschaft f. Chirurgie."

[14] Handbuch der Urologie. A. v. Frisch und O. Zuckerkandl, Band i, p. 651.

[15] Kann der Harnleiterkatheterismus durch Harnsegregatoren ersetzt werden. Berl. klin. Woch., 1903, No. 16.

(2) The membrane should be examined for perforations. (3) The instrument should be sterilized by boiling.

After Using: (1) The membrane should again be examined for perforations. (2) The intravesical portion of the separator should be examined for blood clots. Perforations of the membrane would, of course, negative results. Blood clots on the diaphragm would, as I shall attempt to demonstrate later, modify results.

The Bladder. (1) The bladder capacity should at least be 20 c.c. This limit is given by Lichtenstern. The minimal bladder capacity with which I have used the separator is 50 c.c. When the bladder capacity is much less than this it would seem better to use the smaller Luys instrument, made for children. (2) The urethra should be penetrable to the instrument. (3) Conditions which distort the base or neck of the bladder may prevent separation. The main among these are: (*a*) Marked prostatic hypertrophy; (*b*) extreme anteversion or anteflexion of the uterus; (*c*) various uterine tumors, as advanced carcinoma of the cervix involving the anterior vaginal wall; and (*d*) marked cystocele. (4) The bladder should be cystoscoped. "A classical example for the necessity of this exploration is the perforation of a para- or perimetritic abscess into the bladder, in which with normal kidneys separation alone may lead to the diagnosis of one-sided pyuria." (5) The bladder should be washed with sterile boric-acid solution until the returning fluid is entirely clear, and then 40 to 50 c.c. allowed to remain in the bladder.

Introduction. The separator having been sterilized and lubricated with sterile glycerin, and the bladder prepared under aseptic precautions, the patient is ready for its introduction. The patient should be in the dorsal position, with the trunk horizontal upon the table, the pelvis at the edge of the table, and the feet upon supports below the table level.

With women the introduction is very simple and as a rule painless. The tip of the separator is inserted into the urethra as a sound or catheter, and the instrument following the curve of the urethra glides into the bladder. Vaginal palpation may be employed to verify its position, but I have seldom found this necessary.

With men the introduction is more difficult and more painful. The instrument is introduced into the male urethra in the same manner as a Béniqué sound until the tip is at the prostatic urethra. Then slowly and with much care the handle of the instrument is depressed, there being at the same time a constant and steady pressure along the axis of the separator. With or without a slight jump the separator enters the bladder, whereupon the handle should be elevated. The separator being within the bladder the patient should be elevated to a sitting position, with his back supported at an angle of about 60 degrees to the table.

The diaphragm should be adjusted and the handle of the separator gently raised until the intravesical portion meets resistance along the lower bladder wall. When the separation has been completed the diaphragm should be lowered and the instrument removed, reversing the steps of introduction.

With the separator in position the adjusting of the diaphragm causes little or no pain. If the separator is not entirely within the bladder, the elevation of the diaphragm causes trauma of the vesical sphincter. When this is the case much pain is usually caused by screwing up the diaphragm, and bleeding occurs and will become manifest in the urine that flows from the tubes of the separator.

Before the instrument is entirely within the bladder, fluid begins to run irregularly from the tubes.

It has been my custom to stop the flow of boric solution from the tubes until the separator is wholly within the bladder, the diaphragm adjusted, and the patient elevated to the sitting position; these manipulations being less likely to cause trauma to the mucous membrane if fluid be within the bladder.

When the boric solution has run out the separated urine begins to flow. Lichtenstern says: "A safe index for the proper position of the instrument is given by the manner of the urinary flow from the tubes. Normally, in rhythmical intervals, series of drops are excreted corresponding with the contraction of the ureter and the alternating function of the kidneys. If the instrument is not properly placed the urine can either flow in continuous drops, or the tubes excrete nothing, and between them and the separating rubber membrane the urine trickles out."

Slight hemorrhage occurs, in some cases resulting from the bladder muscle grasping the instrument too vigorously and causing trauma of the bladder mucous membrane. This rarely occurs in normal bladders, not infrequently in diseased, and is manifested (1) by small blood clots along the rubber membrane, seen when the instrument is withdrawn; and (2) by a slight and equal increase of the fresh red blood cells in both of the separated specimens.

The exactness and constancy of this second condition has been proved by comparing, in the same case, the separator results with the results of ureteral catheterization. Thus, if we find slight blood clots along the membrane of the withdrawn instrument, we may expect a slight increase of the fresh red blood cells of both sides and must not mistake their origin and make a diagnosis of renal hematuria. I have never found, in these cases of traumatic hemorrhage, that catheterization of the ureters gave more exact results.

Summary of Cases. The instrument has been used in 14 cases, 2 male and 12 female. In 2 of the 14 cases (Cases I and II) there was absence of the kidney on one side. In a third (Case III) partial nephrectomy had been done. In the remaining 11 cases there were 2 (Cases IV and V) of unilateral pyonephrosis; 2 of pyelitis, bilateral (Cases VI and VII); 1 of pyelonephritis, bilateral (Case VIII); 1 case of unilateral nephritis (Case IX); 1 of tuberculous kidney, unilateral (Case X); 3 cases of unilateral hematuria (Cases XI, XII, and XIII), and 1 case of bilateral hematuria (Case XIV).

Five of the 14 cases have been catheterized (Cases III, VIII, X, XI, and XIV). The results in 3 of the 5 catheterized cases (Cases III, VIII, and XI) agree with the separator results. In 1 of the 5 the separator gave probably more exact results than catheterization (Case X), and in the last of the 5 catheterization apparently gave more accurate results than the separator (Case XIV), but the subsequent history leaves this in doubt. 7 of the 14 (Cases I, II, III, IV, V, VIII, and XIV) have been operated upon either before or after separation. In 6 of these cases the results of separation were confirmed by operation. The seventh is Case XIV, referred to above. All of the cases have been cystoscoped. In none of the cases was the separator proved to be inaccurate. In the cases in which operation was not done nor ureteral catheterization performed, the separator results agreed with the symptoms, physical signs, and cystoscopic findings. In 2 of the 14 cases there was a marked difference in the reaction of the urines of the two sides (Cases IV and V). In 9 of the 14 cases there were pathological conditions of the bladder: 4 with chronic cystitis with ulcerations or erosions; 2 with simple chronic cystitis; 1 of tuberculous bladder; 1 of trabecular hypertrophy; 1 simple congestion.

The following are notes of several of the more interesting cases:

Case I.—Edna G., aged twenty-four years. One and one-half years ago nephrectomy was done, in Bellevue Hospital, for left-sided tuberculous kidney. A sinus in the left lumbar region still persists.

Cystoscopic. Bladder capacity 175 c.c. Bladder wall and ureteral openings normal.

Separation. The separator was easily introduced and remained in place thirty minutes. In that time not a drop of urine came from the left tube of the separator. From the right tube 30 c.c. of clear urine flowed in fifteen minutes.

Analysis. Right urine: Clear, light yellow; acid; albumin, slight trace; △ (freezing point) —0.64; sugar, negative. Precipitate (by centrifuge) slight, showing few red blood cells and few leukocytes.

Case II.—Mrs. Rose T., aged thirty-four years. Admitted to New York Hospital, service of Dr. F. W. Murray, March 21, 1906. For the past year patient has had pain more or less continuous in right lumbar region. The urine has been intermittently cloudy and bloody. Loss of flesh and strength. For the past two months, pain and prostration have been much worse. Fever and chills. Urination very frequent. Right kidney was much enlarged and tender. At operation a right-sided pyonephrotic sac was found, incised, and drained. Two months later the patient was cystoscoped and the separator used.

Cystoscopic. Bladder capacity 200 c.c. Moderate degree of trabeculous hypertrophy present. Left ureteral opening normal, urine coming forth. At the position of the right ureteral orifice there is a mass of reddened granulation tissue. No opening seen.

Separation. The separator was easily introduced and remained in place thirty minutes. In that time two or three drops of whitish fluid were excreted from the right tube. The time of excretion of these few drops corresponded with the periodic vigorous contractions of the irritable bladder upon the separator, and were evidently drops of boric-acid solution remaining in the bladder. From the left tube 17 c.c. of urine was collected.

Analysis. Left urine: Slightly cloudy, amber; acid; albumin, moderate trace; sugar, negative; precipitate (by centrifuge) moderate, showing a moderate number of fresh red blood cells, few leukocytes, and a few squamous epithelial cells.

This case is interesting in that the separator gave exact results in a bladder with trabecular hypertrophy.

CASE III.—Mrs. Mary L., aged twenty-nine years. Case of Dr. T. B. Barringer, Jr. Six years ago, following a bullet wound, a left-sided nephrectomy (according to patient) was done at the Harlem Hospital. The cicatrix of the old wound is present in the left lumbar region.

Cystoscopic. Bladder capacity 200 c.c. Bladder and ureteral openings are normal. Both ureteral mouths contracted regularly, the contractions of the left side being at longer intervals than those of the right.

Separation. Separator easily introduced. From the right tube urine began to flow rhythmically, 20.5 c.c. collecting in forty-five minutes. From the left tube no urine appeared up to ten minutes; then, quite irregularly, urine began to flow from the left side, 7.25 c.c. collecting in forty-five minutes.

Analysis. Right urine: Clear, yellow; acid; albumin and sugar negative. Precipitate (by centrifuge) slight, showing few cylindroids.

Left urine: Cloudy, yellow; acid; albumin and sugar negative. Precipitate (by centrifuge) slight, showing rare leukocyte; rare pyriform cell.

2 c.c. of a 4 per cent. solution of indigo-carmine was injected into the gluteal region. In twenty-five minutes the right urine was decidedly blue. On the left side there was no trace of blue up to thirty-five minutes.

At a later date the separator was again used: from the right side 99 c.c. of urine collected in seventy minutes. For thirty-five minutes no urine flowed from the left tube: from then up to seventy minutes 3.75 c.c. of urine was collected. The patient's ureters were now catheterized. The right ureter was easily catheterized; the left ureter admitted only a small catheter. In fifteen minutes 65 c.c. of normal urine was collected from the right. Up to thirty minutes none was secreted from the left.

In this case while the separator results do not entirely correspond with catheterization, there are two reasons for believing that the former was exact: (1) Cystoscopically, the left ureteral mouth was seen to contract more or less rhythmically; this would not be if it had not excreted urine for six years. (2) After the indigo-carmine injection for ten minutes there flowed a fairly intense blue from the right tube of the separator and absolutely no trace of color from the left side. This would be impossible if fluid from the left side was the result of leakage from the right.

Case IV.—Mrs. Sarah M., aged thirty-five years. Case of Dr. E. D. Barringer.[16] For past year pain in right lumbar region, cloudy urine, frequent urination, progressive loss of flesh and strength. The right kidney was enlarged and tender.

Cystoscopic. Bladder capacity, 100 c.c. Urine very cloudy. Right ureteral orifice swollen, ulcerated. While being observed a thick, dense, yellow stream of pus was excreted from ureteral orifice. Left ureteral orifice, mucous membrane slightly swollen; clear urine excreted.

Separation. The separator was easily introduced. In eight minutes 5 c.c. of thick yellow pus flowed irregularly from the right tube, and 49 c.c. of clear urine from the left.

Analysis. Right urine: Opaque, yellow; strongly acid; albumin, heavy trace; sugar, negative. Half of the fluid is precipitate composed almost entirely of pus cells; rare red blood cell.

Left urine: Clear, yellow, slightly acid; albumin, negative; sugar, negative. Precipitate (by centrifuge) slight, composed of occasional red blood cells, leukocytes (no pus), and epithelial cells.

Diagnosis. Right pyonephrosis. The patient was operated upon at Mount Sinai Hospital where a right pyonephrosis with multiple stones was found.

The difference in reaction of the urine from the two kidneys is of interest.

Case V.—Mrs. Ada M., aged thirty-three years. Admitted to New York Hospital, February 16, 1906, in the service of Dr. F. W. Murray. The patient was operated upon by Dr. Murray, who found in the left kidney a number of large abscess cavities with multiple stones. The stones were removed and the kidney drained. Some weeks later the patient was cystoscoped and the urine separated.

Cystoscopic. Bladder capacity, 100 c.c. Urine turbid with pus. Pus was being excreted into bladder so rapidly as to make the cystoscopic examination difficult, and it would have been hard if not impossible to catheterize the ureters. Pus was seen, thick and yellow, coming from the left ureter. The right ureteral mouth was imperfectly seen: chronic cystitis of base of bladder.

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[16] This case was separated and cystoscoped by Dr. E. D. Barringer.

Separation. The separator was introduced with considerable pain, without rewashing the bladder. Thick yellow pus began to flow, drop by drop, from the left tube: 3 c.c. in fifteen minutes. Slightly cloudy urine flowed rhythmically from the right: 10 c.c. in fifteen minutes.

Analysis. Right urine: Slightly turbid, amber; acid; albumin, trace; sugar, negative; urea, 0.003 gram to c.c. Precipitate (by centrifuge) moderate; composed of few red blood cells, many pus cells, and a moderate number of round and squamous epithelium.

Left urine: Opaque, yellowish white; alkaline reaction; albumin, marked trace; sugar, negative. One-half of the fluid was precipitate, composed almost entirely of pus cells, few round epithelial cells.

Diagnosis. Left, pyonephrosis; right, pyelitis. Three days later the separator was again used, the bladder being washed entirely clean. The bladder urine showed much less pus and a much larger discharge came from the nephrotomy incision.

In twenty-five minutes but 1 c.c. of turbid fluid flowed from the left tube. From the right, 20 c.c. of nearly clear urine flowed. The analysis of these urines was the same as above, the only difference being in the quantity of pus excreted from the left side. 1 c.c. of a 1 per cent. phloridzin solution was injected into the buttock; sugar appeared in the right urine in from thirty to thirty-five minutes, and none in the left urine. At a second operation the left kidney was found practically composed of abscess cavities.

Case VI.—Mrs. Fannie M., aged thirty years. Admitted to New York Hospital, in the service of Dr. Bolton, September 11, 1905. Patient's history suggests attacks of intermittent hydronephrosis. While in the hospital she had several chills and fever. Bilateral nephroptosis, much more marked on right, was present.

Cystoscopic. Bladder capacity 175 c.c. Entire bladder with ureteral openings congested. Urine coming from both ureteral orifices.

Separation. Separator easily introduced, urine flowing rhythmically from both tubes, 12 c.c. in thirty minutes, from right; 11 c.c. in thirty minutes, from left.

Analysis. Right urine: Clear, yellow; reaction, acid; albumin, marked trace; sugar, negative; △ —0.91. Precipitate (by centrifuge) slight, showing few red blood cells; many leukocytes; pus; few pyriform epithelium; few cylindroids.

Left urine: Clear, yellow; reaction, acid; albumin, marked trace; sugar, negative; △ —1.62. Microscopically, the same as right.

3.5 c.c. of a 4 per cent. solution of indigo-carmine was injected into the gluteal region with the following results: in twenty-three minutes marked blue from left side; right urine, no trace of blue;

in thirty minutes, distinct blue on right side, never as marked as on the left.

Diagnosis. Bilateral pyelitis. Decreased functional activity of the right kidney as compared with left. This latter finding corresponded with the history, that of intermittent hydronephrosis, and with the physical examination.

Case VII.—Mrs. D. M., aged twenty-nine years. Case of Dr. T. B. Barringer, Jr. Some weeks before being seen the patient developed bilateral pyelitis, following cystitis. She then developed symptoms pointing toward obstruction of the left ureter. She was seen with a view to ascertaining if the left ureter was plugged, and if so, to attempt to catheterize it to drain off the pus. Both kidneys were markedly tender, and the right kidney decidedly enlarged.

Cystoscopic. Bladder capacity, 200 c.c. The base of the bladder showed a marked cystitis with eroded areas. Ureteral openings could not be seen.

Separation. Separator introduced without pain. Slightly cloudy urine flowed rhythmically from both tubes: 6.5 c.c. in ten minutes from right; 6.5 c.c. in ten minutes from the left.

Analysis. Right urine: Slightly cloudy, yellow, acid; albumin, heavy trace; sugar, negative; △ —1.09.

The moderate precipitate (by centrifuge) was composed almost entirely of cells of the renal collecting tubules and pelvis of the kidney with occasional leukocytes and red blood cells.

Left urine: The analysis and microscopic examination corresponded with the right, △ being —0.91.

Diagnosis. Bilateral pyelitis. In this case, because the ureteral openings could not be seen, the only way to diagnose positively the permeability or impermeability of the left ureter, without the use of the separator, was to await the development of symptoms.

Case IX.—Dora T. H., aged fifteen years. Patient in Dr. A. B. Johnson's service, New York Hospital. Patient gave a history pointing to disease of the left kidney.

Status Localis. Tenderness and rigidity in right lumbar region. The analysis of a non-catheterized specimen of bladder contents was as follows: A turbid, amber, acid urine; moderate flocculent precipitate; specific gravity, 1026; albumin, very faint trace; sugar, negative. Much pus; hyaline, granular and leukocytic casts; few epithelial cells. The presence of hyaline and granular casts in the patient's urine was constant.

Cystoscopic. Bladder and ureteral openings normal.

Separation. Urine flowed rhythmically from both tubes, 9 c.c. in twenty minutes from right, and 10 c.c. in twenty minutes from left.

Analysis. Right urine: Clear, yellow, acid; albumin, trace; sugar, negative; △ —0.94. Very slight precipitate composed of rare leukocyte, frequent hyaline and granular casts.

Left urine: Clear, yellow, acid; albumin, negative; sugar, negative; △ —1.34. Microscopic, negative.

On a later separation both urines were normal. Later a profuse vaginal discharge made its appearance. An exploratory laparotomy showed large right-sided pyosalpinx.

Diagnosis. Because of the constant appearance of albumin and casts in the bladder urine, and because in two separated specimens the left urine was normal, a diagnosis of right-sided nephritis was made, caused possibly by the right pyosalpinx.

Case X.—Hazel T., aged fourteen years. Admitted to service of Dr. F. W. Murray, New York Hospital, April 18, 1906.

History covering eighteen months of increased frequency of urination and cloudy urine. Deep pressure gives tenderness over the right kidney. Ureteral catheterization was performed: 15 c.c. flowing from the right side in ten minutes, and 15 c.c. from the left in ten minutes.

Analysis. Right urine: Turbid, yellow, acid; albumin, trace; sugar, negative. Urea 0.002 gram to c.c. Moderate precipitate (by centrifuge), showing many red blood cells; many pus cells; moderate number of renal and ureteral cells; one hyaline cast; many tubercle bacilli.

Left urine: Turbid, yellow, acid; albumin, trace; sugar, negative. Urea, 0.002 gram to c.c. Microscopic, was the same as the right, with the exception that no tubercle bacilli were found. The only difference in the two sides is the finding of tubercle bacilli in the right in considerable numbers, and the failure to find them in the left. A few days later the patient was cystoscoped and urine separated.

Cystoscopic. Bladder capacity, 50 c.c. Right ureteral orifice enlarged, reddened, and thickened. Mucous membrane swollen around ureteral mouth and ulcerated in places. Left ureteral mouth normal.

Separation. The separator was introduced with considerable pain. In twelve minutes 4 c.c. of turbid urine flowed from right side, irregularly, and 20 c.c. of clear urine flowed rhythmically from the left.

Analysis. Right urine: Cloudy, yellow. (Albumin, etc., not taken.) Marked precipitate (by centrifuge) composed of many blood shadows; some fresh red blood cells; many pus cells; much renal epithelium; occasional hyaline and granular casts; many tubercle bacilli.

Left urine: Clear, yellow. (Albumin, etc., not taken.) Slight precipitate (by centrifuge) composed of very many fresh red blood cells; occasional leukocyte, no pus; occasional renal and vesical epithelium; one epithelial, two hyaline casts; no tubercle bacilli.

This was a case in which catheterization of both ureters was very difficult and the separator apparently gave more accurate results. The left ureteral orifice was by cystoscopic examination entirely normal. The main abnormal finding of the separated left urine was "many fresh red blood cells." But these were the precipitate of 20 c.c. of fluid, while there was but 4 c.c. of right urine to give the precipitate for microscopic examination. So, relatively, there must have been but few red blood cells in the left urine, and these were traumatic, the bladder being small, irritable, and tuberculous. No pus was found in the left urine. A few hyaline and epithelial casts were present, and there was a marked difference in the amount of urine secreted: 4 c.c. from the right as compared with 20 c.c. from the left, in twelve minutes.

Diagnosis. Right tuberculous kidney, normal left kidney. The patient is expected to come to operation.

CASE XIV.—James J. M., aged twenty-four years. For past ten years he has had attacks of pain in left kidney region, sharp and stabbing, running into left testicle. Over the left kidney and along the course of the left ureter there is rigidity and tenderness.

Cystoscopic. Bladder normal; left ureteral orifice slightly congested.

Separation. Separator introduced, with some pain—none when in position. Urine flowed rhythmically from right side, 9 c.c. in thirty minutes; irregularly from left, 12 c.c. in thirty minutes.

Analysis. Right urine: Clear, amber, acid; albumin, heavy trace; sugar, negative. Moderate precipitate (by centrifuge) composed of many red blood cells; many leukocytes, pus; many squamous; few pyriform cells.

Left urine: Exactly the same as the right.

From the separation, it was thought, notwithstanding that all the symptoms pointed toward the left kidney as the main seat of the trouble, that the right kidney was also affected.

The patient was admitted to the New York Hospital, where the ureters were catheterized, with the following results:

Analysis. Right urine: Clear, amber, acid; albumin, faintest possible trace; sugar, negative. Slight precipitate (by centrifuge) composed of occasional red blood cell, occasional leukocyte; numerous spheroid, and caudate epithelium.

Left urine: Clear, light amber, acid; albumin, faint trace; sugar, negative. Moderate precipitate (by centrifuge) composed of many red blood cells; many pus cells; occasional renal cell; no tubercle bacilli.

The patient was operated upon by Dr. Markoe, who removed a stone from the left kidney pelvis. The catheterized specimen apparently gave more accurate results than the separated. Both gave abnormal findings for the right kidney, but the separator much more positively so. The separation took place just after the patient had walked to my office; the catheterization, after he had been in bed two days. When seen about three months after leaving the hospital he gave a history of symptoms of the right kidney, pain, etc., exactly similar to those he had on the left side. Since then the patient has not been seen.

Conclusions. 1. There is a large class of cases in which knowledge of the condition of the kidneys is necessary and in which the ureters cannot be catheterized. These are cases in which (*a*) one or both ureteral openings are obscured by cystitis; (*b*) in which the rapid excretion of pus or blood into the bladder clouds the bladder fluid; or (*c*) in which in a normal bladder one or both ureteral mouths cannot be found. These are cases in which the separator is invaluable.

2. Cystitis forms no barrier to the use of the separator and to obtaining an exact knowledge of the kidney conditions in such cases. The exception to this is when a bacteriological examination is necessary, when catheterization of the ureters is more accurate.

3. Separation is much simpler than ureteral catheterization. The sterilization of the instrument is absolute, as the separator can be boiled. Infection of the ureters from the bladder is excluded.

4. In women the pain or discomfort caused by the separator is about the same as that caused by the use of a simple examining cystoscope. In men the pain of separation is slightly more marked.

5. There are certain classes of cases in which the separator cannot be used. They are the following: (A) Those in which the bladder capacity is less than 20 c.c.; (B) those in which the urethra is not penetrable to the instrument; and (C) those in which the base or neck of the bladder is distorted by (*a*) marked prostatic hypertrophy; (*b*) extreme anteversion or anteflexion of the uterus; (*c*) certain uterine tumors; and (*d*) marked cystocele.

6. Slight hemorrhage occurs in some cases resulting from the bladder muscle grasping the instrument too vigorously and causing trauma of the vesical mucous membrane. This is shown by (*a*) small blood-clots along the membrane when the separator is withdrawn; and (*b*) slight and equal increase in the fresh red blood cells of both sides.

7. Any inaccuracy of results obtained by the use of the separator is to be attributed to its employment in unsuitable cases or to a failure to recognize traumatic hemorrhage when it occurs; and does not arise from leakage of the urine from one side to the other.

I wish to express my thanks to Dr. Samuel Alexander for many valuable suggestions; to Drs. F. W. Murray, P. R. Bolton, A. B. Johnson, and F. H. Markoe for the use of cases in their respective services at the New York Hospital; to Drs. T. B. Barringer, Jr., C. E. S. Webster, Jr., and E. D. Barringer for referred cases; and to Drs. J. C. Roper, T. W. Hastings, and Mortimer Warren for many urine analyses.

THE COLLOIDAL NITROGEN IN THE URINE.

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In the course of an investigation[1] which one of us has been making into the clinical significance of the complete analysis of the urine with respect to the nitrogenous constituents, our attention has been called to a paper by E. Salkowski[2] on a fraction of the nitrogen which is precipitated by absolute alcohol. This fraction he terms colloidal nitrogen.

It would appear from Salkowski's paper that in speaking of colloidal nitrogen, he had in mind the large number of substances which are comprised under the head of undetermined nitrogen, that nitrogen which is left unestimated after the more prominent constituents of the urine, the urea, ammonia, uric acid, creatinin, and creatin are estimated. The nature of these substances is not well understood, but among them are the sulphur-containing acids, the proteic acids which have been recently made the subject of renewed investigation by Bondzynski[3] and his co-workers.

From a clinical point of view, one thing seems probable: The undetermined fraction is increased both absolutely and relatively to the undetermined nitrogen in many metabolic disturbances of pathological origin.

As the methods for estimating the undetermined nitrogen are outside the range of clinical technique, and the method for estimating the colloidal nitrogen could be performed by the clinician without a great deal of difficulty, it appeared desirable to correlate the two methods in order to ascertain whether they bear a relation to one another. Furthermore, the results obtained by Salkowski in the analyses of normal and pathological urine bore a striking similarity to some of our own which were made with the more exact methods of urine analysis.

At the outset, it must be confessed, it did not seem probable that the two methods would agree, for part of the substances precipitated by alcohol are present in the determined nitrogen, while some of the compounds not precipitated by alcohol are members of the class of undetermined nitrogen. In the former class would be included creatin and creatinin and the urates, while in the latter would be included the alcohol-soluble amino-acids, glycocoll and leucin.

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[1] Ewing and Wolf. Amer. Jour. Med. Sci., 1906, cxxxi, 751.

[2] Berl. klin. Woch., 1905, xlii, 1581, 1618.

[3] Bondzynski, Dombrowski, and Panek. Ztschr. f. physiol. Chem., 1905, xlvi, 83.

As glycocoll has recently been made the subject of a number of investigations[4] which tend to show that either the acid itself or a compound in which the acid is an integral part is a constituent of normal urine in conspicuous amount, and as this finding in its clinical relation is at present the subject of an interesting discussion, our interest in the matter of colloidal nitrogen was increased. If it could be shown that the amount of glycocoll is an important feature in the analysis of the urine, we should expect to find that the colloidal and undetermined nitrogens from this cause alone would not be in close agreement.

The methods used in the determination of the urea, ammonia, uric acid, creatinin, creatin have been detailed by one of us in a recent paper.[5] The estimation of colloidal nitrogen is a simple procedure, and when properly carried out yields closely agreeing duplicate analyses. One hundred c.c. of urine is evaporated slowly to a syrup. To the residue 100 c.c. of absolute alcohol is added, and the mixture allowed to stand for a few minutes. The concentration of the urine should be regulated carefully. If the urine is too concentrated, the addition of alcohol precipitates a gummy mass, which cannot be washed free from urea. If the urine is not concentrated sufficiently, the colloidal nitrogen is incompletely precipitated. It is also advisable to add the alcohol with constant stirring while the residue is quite hot. Under these circumstances, the colloidal nitrogen is precipitated in a flocculent condition, is easily transferred to a filter, and should be washed with small quantities of absolute alcohol.

The following table gives the results which were obtained with eleven samples of urine. The specimens were chosen to represent as varied conditions as possible.

No.	T. N	$\frac{\text{U. N.}}{\text{T. N.}}$	$\frac{\text{NH}_3\text{.N.}}{\text{T. N.}}$	$\frac{\text{Undt. N.}}{\text{T. N.}}$	$\frac{\text{Coll.N.}}{\text{T. N.}}$	Undt.N. g.	Coll. N. g.	$\frac{\text{Coll. N.}}{\text{Undt.N.}}$	
166	4.3	68.8	7.2	17.3	9.3	0.744	0.406	54.3	Hyperemesis of pregnancy.
170	5.4	59.7	8.1	21.8	13.2	1.170	0.691	60.0	Pregnancy.
179	11.8	81.8	5.2	8.3	5.2	0.980	0.620	62.8	
180	8.1	77.4	4.3	11.8	11.2	0.960	0.920	95.5	Dermatitis herpetiformis.
201	5.5	73.3	2.4	18.0	12.1	1.010	0.670	67.0	Pregnancy. Normal.
239	5.5	82.0	5.0	7.9	5.2	0.440	0.290	65.8	Leukemia in boy aged eleven years.
240	25.0	85.4	5.4	3.9	4.0	0.730	0.750	103.0	Diabetes. Strict diet. No glucose.
242	8.3	79.5	2.4	13.4	10.0	1.940	1.440	74.5	Antepartem.
250	12.0	81.1	5.8	7.2	5.0	0.870	0.600	69.0	Herpes.
251	4.7	72.8	9.4	10.9	7.2	0.510	0.340	66.0	Eclampsia.
252	6.4	72.4	7.7	12.5	7.0	0.820	0.450	54.9	Pregnancy 8th month. Œdema.

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[4] Samuely. Ztschr. f. physiol. Chem., 1906, xlvii, 376.

[5] Ewing and Wolf, loc. cit.

The net result of the examination is given in the last column, where the relation of the colloidal nitrogen to the undetermined nitrogen has been worked out. It will be seen that the relation is in no sense a constant one, but varies from 54 per cent. to 103 per cent. In the majority of the specimens the colloidal nitrogen forms about 60 per cent. of the undetermined nitrogen, but in one instance, in which the patient, a diabetic, was consuming large amounts of proteins, the colloidal nitrogen was present in larger amount than the undetermined nitrogen.

It would seem that the variation is much too great to permit one to use the method, even for clinical purposes, either as an equivalent or substitute for the more accurate methods for estimating the undetermined nitrogen.

In the second part of the paper on colloidal nitrogen Salkowski made an examination of the properties of the mixture which is precipitated by alcohol. Among the substances which were found was a nitrogen-containing carbohydrate which did not reduce Fehling's solution, except after inversion with dilute acids or after treatment with ptyalin. In order to confirm Salkowski's interesting observation, we prepared a quantity of the colloidal nitrogen, and subjected it to an examination.

For this purpose, 28 liters of normal urine was evaporated gently on an electric stove, and to the hot residue 5 liters of alcohol was added. The mixture was allowed to stand over night. The residue, consisting chiefly of inorganic salts, was filtered off. The filtrate was evaporated to a syrup, and reprecipitated with a large volume of absolute alcohol. The precipitate was again dissolved in water, evaporated, and precipitated for the third time. The precipitate was collected on a filter, washed with a large quantity of absolute alcohol, and dried in a vacuum. The vacuum-dried residue weighed 2.2 grams. It formed a dark-brown powder which was completely soluble in water.

A qualitative examination of the mixture dissolved in water gave the following results: Xanthoproteic reaction, slight. Biuret reaction, negative. Millon's reaction, brown or reddish-brown precipitate. Adamkiewicz reaction, negative. Picric acid, slight cloudiness. Potassium ferrocyanide and acetic acid, no precipitate. Mercuric chloride, cloudiness. Voluminous red-brown precipitate on heating. Mercurous nitrate, red precipitate in the cold. Lead acetate, brown flocculent precipitate, soluble in acetic acid. Cupric acetate, brown precipitate, soluble in acetic acid. Orthophosphoric acid, 50 per cent., no precipitate. Phosphotungstic and hydrochloric acid, brown voluminous precipitate. Tannic acid, faint precipitate. Ferric ammonium sulphate and sulphuric acid, faint precipitate. Phosphomolybdic acid, brown precipitate. Alkaline lead acetate and heat, no darkening. Ammonio-silver nitrate and heat, no reduction. Molisch's test, exceedingly faint. Haines' solution, no reduction. Haines' solution after heating solution with hydrochloric acid, no reduction.

The determination of nitrogen and ash in the substances gave the following results: Determination of ash: 0.0278 gram of the substance dried at 110° gave, on ignition, 0.0262 gram of ash = 9.4 per cent. Nitrogen determined by Kjeldahl = 8.5 per cent. Amount of nitrogen in ash-free substance = 9.6 per cent.

As unquestionably a part of the substances which are precipitated by alcohol are the sulphur-containing proteic acids above referred to, it was thought of interest to determine the partition of the sulphur in the substance directly. A portion of the substance was dissolved in water, and the total sulphur, total sulphates, alkaline sulphates, ethereal sulphates, and neutral sulphur were estimated. The analyses gave the following results: Partition of sulphur in the substance: Total sulphur, 1.65 per cent. Total sulphate sulphur = 0.46 per cent., = 27.8 per cent. of total sulphur. Alkaline sulphate sulphur = 0.022 per cent., = 1.33 per cent. of total sulphur. Ethereal sulphate sulphur = 0.44 per cent., = 26.5 per cent. of total sulphur. Neutral sulphur = 1.19 per cent., = 72.2 per cent. of total sulphur.

It will be observed that the greater portion of the sulphur present is in the form of neutral sulphur, to which the proteic acids belong. A not inconsiderable portion is represented by ethereal sulphates, comprising perhaps indoxyl and phenolic sulphates. The amount of inorganic sulphates is about 1 per cent. of the total.

It is interesting to note that the percentage of neutral sulphur does not allow one to form a judgment of the amount of proteic acids present in the colloidal nitrogen. On the one hand, the percentage of neutral sulphur in the substance is, as a whole, greater than the average of the sulphur analyses of the compounds as found by Bondzynski, and the nitrogen content of the colloidal nitrogen as found by us is much lower. One can only conclude, therefore, that in the colloidal nitrogen mixture there are other sulphur-containing compounds which have a much lower sulphur content than the class of proteic acids. What the nature of these compounds is one cannot even conjecture. An attempt was also made by Salkowski to effect a partial separation of the mixture contained in the colloidal nitrogen by means of animal charcoal. We have also repeated his experiments in this direction.

Separation by means of animal charcoal: 0.6 gram of the substance was dissolved in 100 c.c. of water and 5 grams of Kahlbaum's "K" animal charcoal added. The mixture was heated over night on the water-bath and filtered. The filtrate was colorless. The filtrate was tested by the following tests: Molisch's test, faint violet color. Ammoniacal silver nitrate and heat, no mirror. Haines' solution, no reduction. Haines' solution after hydrochloric acid and heat, no reduction. Haines' solution after ten hours in thermostat at 40° with saliva, no reduction. Iodopotassium iodide, no color change or precipitate. Tannic acid, negative. Murexide test, negative. Biuret test, negative. Lead acetate, white flocculent precipitate on warming.

Analysis of the filtrate for nitrogen and ash: 25 c.c. of solution evaporated and dried at 110° gave 0.0324 gram of residue. Substance gave on ignition 0.0116 gram of ash = 35.8 per cent. Nitrogen in dried residue = 11.7 per cent. = 18.2 per cent. in ash-free substance.

The animal charcoal was extracted with ammoniacal 50 per cent. alcohol. A thick brown liquid was obtained, which with zinc and hydrochloric acid evolved hydrogen sulphide.

In regard to the nitrogenous carbohydrate of Salkowski, it will be seen that we have not been able to confirm his results. While the presence of a carbohydrate group is possible from the faint Molisch test which we obtained, this test is too delicate to conclude that the substance is really a definite part of the colloidal nitrogen. As far as a reducing carbohydrate, which is to be obtained after inversion, is concerned, the tests which were designed to show its presence absolutely failed.

SUMMARY. The colloidal nitrogen of Salkowski does not bear any relation to the undetermined nitrogen. The method cannot be recommended for clinical use. In the mixture precipitated by alcohol from urine the nitrogenous carbohydrate described by Salkowski was not found.

A COMPARATIVE STUDY OF THE OCCULT BLOOD TESTS; A NEW MODIFICATION OF THE GUAIAC REACTION; ITS VALUE IN LEGAL MEDICINE.

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OCCULT blood reactions, chemical reactions for the recognition of minimal hemorrhages into the gastrointestinal tract unrecognizable by the unaided eye, have been the subject of considerable discussion among internists during the last two or three years. It has been but a very short time since much stress has been laid upon the value in the differential diagnosis of gastrointestinal diseases of the recognition of these minimal hemorrhages. Our present knowledge of the subject may be said to date from the appearance of Boas' first article.[1] So little attention was given to this that in his second article which appeared a year later, he expresses surprise that clinicians have neglected what he considers a great advance in our clinical knowledge of these diseases. Practically two years passed before the work was seriously taken up by others, and then followed contributions from Schmilinsky,[2] Joachim, Schloss,[3] Hartmann,[4] Clemm,[5] and a few others, all of whom reassure us of the value of these tests. The history of the original Weber[6] reaction has been one of repeated modifications and a changing of the conception of what is and what is not a proper end reaction.

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[1] Sammlung klin. Vorträge, February, 1905, Nr. 387. (Many other references.)

The tests fortunately are very simple, but experience has taught us that this simplicity must be conducted with decided care, particularly in experimental work. Weber's test and all of its modifications depend upon three definite principles: (1) That changed hemoglobin is soluble in acetic acid, forming acid hematin; (2) that acetic acid solutions of hematin are soluble in sulphuric ether; and (3) that guaiac resin when dissolved and ozonized in a solution of blood causes a typical color reaction. I give herewith the original Weber test and its most important modifications, as well as the Klunge aloin test. For convenience the successive steps in each test have been numbered.

Weber's Test. 1. Five c.c. of stomach contents or a similar amount of softened stool is mixed with one-third its amount of glacial acetic acid.

2. To the above mixture add an excess of ether and allow to extract.

3. To a portion of the resulting extract add ten drops of a freshly prepared tincture of guaiac, and shake thoroughly.

4. Add thirty drops of old turpentine.

If the reaction is positive an intense blue color results. If there is much blood present the ether extract becomes a Tokay color. If there is little blood the ether remains uncolored.

Boas' First Modification of the Weber Test. 1. About 5 c.c. of soft stool is extracted with 20 c.c. of ether to remove the fats.

2. Pour off the resulting extract.

3. Add 2 or 3 c.c. of glacial acetic acid to the remaining stool, and mix thoroughly.

4. Extract with 10 c.c. of ether.

5. Add a few granules of powdered guaiac resin to a portion of the above extract in a test-tube and shake carefully.

6. Add thirty drops of old turpentine.

The main difference is that Boas first extracts with ether to remove the fats that may be present and employs the powdered guaiac in place of the tincture.

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[2] Münch. med. Woch., 1903, Nr. 49.

[3] Arch. f. Verdauungskrankheiten, Band x, Heft 3.

[4] Ibid., Band x, Heft 1.

[5] Ibid., Band x, Heft 4.

[6] Berl. klin. Woch., 1893. No. 19.

Boas' Second Modification of the Weber Test. Instead of using old turpentine Boas later recommended the use of dilute hydrogen peroxide—a 3 to 5 per cent. solution. He was then of the opinion that it was not necessary first to remove the fats, and this part of the test was dispensed with. Boas found this method more delicate than the turpentine method.

Rossel advises that when the test is applied to the stomach fluid it should first be neutralized. Boas claims that neutralization is not necessary. This is very easy to demonstrate.

Author's Water Modification. 1. All glassware must be chemically clean and perfectly dry.[7]

2. To one gram of stool which has been softened if necessary by as few drops of water as possible, about 4 or 5 c.c. of glacial acetic acid is added and intimately mixed. This is best accomplished by rubbing up in a glass mortar.

3. To the above mixture add 30 c.c. of ether and allow to extract for several minutes, after thoroughly shaking or mixing.

4. To 1 or 2 c.c. of the extract, which is perfectly clear and can be decanted off, add an equal amount of distilled water and shake thoroughly.

5. To the resulting mixture add a few granules of powdered guaiac resin, an amount that will be held on the tip of a small knife-blade; this is allowed to dissolve and the tube is agitated thoroughly.

6. Thirty drops of old water-white chemically pure turpentine is now added, and thoroughly mixed with the contents of the tube. Set aside against the light or in front of a white surface.

If blood is present to the amount of 1 milligram to 1 gram of stool a distinct light-blue color develops quickly in the upper half of the mixture, remains for a short time, and gradually disappears. Larger amounts of blood cause a more intense blue, but almost always a turbid, or opaque blue, often the color of a turquoise.

Note.—If the reaction is intense it may be better seen if about 10 c.c. of ether is added to the original extract. If smaller amounts of blood are present the reaction may assume a smoky grass-green color. (See later.) The reaction may be sometimes intensified by extracting the greater part of the coloring matter with alcohol which may be filtered off. This makes the test long and it is seldom necessary.

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[7] The precaution of absolute dryness is not necessary if comparatively large amounts of blood are present. My assistant, Dr. Roche, obtained a positive blue in the early morning several times by the author's method. Other methods were negative. I was unable to verify the results at 11 A.M., neither could Dr. Roche. We then sought for an explanation. Fearing that the early morning positive reaction was due to contamination of the tubes they were all washed with nitrohydrochloric acid and thoroughly rinsed in distilled water. No reaction occurred in these tubes An old tube that had not been used and which did not look clean was now used and a beautiful blue reaction occurred. New tubes that had not been unwrapped also permitted the reaction to take place. We repeated this experiment many times, always obtaining a reaction in a perfectly dry tube, never obtaining it in a damp tube. The other modifications did not take place in the dry tubes. From this time on a set of tubes was kept thoroughly dry by draining over night and heating in the oven if necessary. The reason for the reaction occurring in the early morning was due to the fact that the tubes had dried over night.

Several other modifications might be mentioned, but on careful perusal they will be found to be unintentional modifications, due to a misconception of the successive steps of the original reaction; when applied to very minute amounts of blood they do not react. As the re-extraction with ether has been shown by Boas and others, and can be easily demonstrated, to be of no importance, it is omitted in the experiments that follow; the difference between the two tests being simply in the oxidizing agent employed. Other variations that have been made are noted.

Aloin Test. Klunge has introduced what is known as the aloin test, the successive steps of which are as follows, the principle of the reaction being the same as the guaiac reaction:

To the acetic acid and ether extract obtained as above, 20 to 30 drops of ozonized turpentine is added. The resulting mixture is now treated with from 10 to 15 drops of a freshly prepared tincture of aloin. (About 3 grains to 5 c.c. of 70 per cent. alcohol.) If blood is present, a beautiful cherry-red color gradually develops, but sometimes requires many minutes. By adding a few drops of chloroform the color change may be hastened and intensified. The cherry-red color usually develops in the bottom layer, which is the aloin layer. At other times the entire contents of the tube are changed to a pink or light cherry color. A contact test results if the aloin solution is allowed to run down the side of the tube. A slight pink color will often develop in an hour's time in the absence of blood. The reaction should occur within one-half hour to be positive. Boas employs this test as confirmatory of the guaiac test. He thinks that at times it is more sensitive than the guaiac test.

EXPERIMENTS.

Series I.—Meat Diet Investigations.

It has been claimed that reactions for occult blood occur in the stools of individuals eating a meat diet, and that for this reason the test should be applied only when the patient is eating a meat-free diet. The purpose of this experiment is, therefore, to inquire into the frequency with which the reactions do occur with the ordinary meal. To this end patients were selected without any reference to their ailment; they were eating the regular hospital diet, which consists of meat at least twice and often three times daily. In an institution where hundreds are being fed the meat is not always thoroughly cooked, that is, rare meat is commonly seen. The conditions, accordingly are well adapted for this experiment.

In all, the stools of thirty-four patients were examined. The consistency varied from partly formed to those containing hard and dry cylinders. In only two cases were positive results obtained (Cases II and XX). Inquiry disclosed the fact that Case II had had a visible hemorrhage the previous day, and Case XX, in whom there had been no suspicion of hemorrhage, was found on subsequent examination to have postnasal conditions which caused a more or less constant oozing into the digestive tract.

Both the guaiac and the aloin tests were made in each case. The experiment illustrates that the tests are not delicate enough to recognize the small amounts of blood that are swallowed with the uncooked portions of the meat. It can be said that individuals on an ordinary meat diet seldom if ever give a reaction for occult blood, and that if a reaction occurs it is in all probability due to some pathological process in connection with the gastrointestinal canal. In this series the ether extract was very little influenced by the color of the stool.

Since the above tests were made we have tested a large number of stools of patients under the same conditions, and have yet to find a positive reaction due to uncooked meat.

Series II.—Meat Ingestion Experiments.

The following experiments were made in order to gain some idea of the amount of uncooked red meat that must be ingested before a reaction for occult blood can be obtained in the stools, and also to determine the length of time after the ingestion of the meat before the reaction appears and disappears. The meat was prepared by scraping round steak and chopping into it a very small amount of onion, pepper, and salt. No fiber was present. The amounts were weighed on the ordinary laboratory balance, and are of course only approximate, yet are fairly accurate. The method that has been found the most delicate is the one that is recorded.

EXPERIMENT 1.—Mrs. G. 50 grams of scraped beef was given at 11 A.M.; first stool passed in twenty-one hours; water, guaiac, turpentine method was positive; Klunge test was negative. Second stool was not saved.

EXPERIMENT 2.—50 grams of scraped beef was given at 11 A.M. First stool passed in twenty hours; water, guaiac, turpentine method was positive; Klunge aloin test was negative. Second stool passed in forty-two hours; water, guaiac, turpentine method was negative; Klunge aloin test was negative. Third stool passed in seventy-two hours; water, guaiac, turpentine method was negative; Klunge test was negative.

EXPERIMENT 3.—40 grams of scraped beef was given at 11 A.M. First stool passed in twenty-one and a half hours; water, guaiac, turpentine method was positive; Klunge test was negative. Second stool passed in forty hours; water, guaiac, turpentine method, and Klunge test were negative. Third stool passed in seventy hours; all tests were negative.

EXPERIMENT 4.—Mrs. E. 30 grams of scraped beef was given at 11 A.M. First stool passed in twenty-one hours; water, guaiac, turpentine method was positive; Klunge aloin test was negative. Second stool passed in seventy hours; water, guaiac, turpentine method, and Klunge test were negative.

EXPERIMENT 5.—Mr. E. 20 grams of scraped beef was given at 11 A.M. First stool passed in ten hours; water, guaiac, turpentine method was positive;[8] Klunge aloin test was negative.

EXPERIMENT 6.—Mr. D. 15 grams of scraped beef was given at 11 A.M. First stool passed in twenty hours; water, guaiac, turpentine method was questionable; Klunge aloin test was negative.

EXPERIMENT 7.—Mrs. G. 10 grams of scraped beef was given at 11 A.M. First stool passed in nine and a half hours; water, guaiac, turpentine method was negative; Klunge aloin test was negative. Second stool passed in twenty-four hours; water, guaiac, turpentine method, and Klunge test were negative.

EXPERIMENT 8.—Mr H. Stomach was thoroughly washed and drained. At 11 A.M. 10 grams of chopped beef was given. The first stool was passed in eighteen hours. Hard, brownish-green cylinders one inch in diameter, with no visible blood. Extract, water, guaiac, turpentine method, change to blue-green which faded; extract, guaiac, turpentine method was negative; extract, guaiac, dilute H_2O_2 method, black reaction in upper layer; extract, guaiac, strong H_2O_2 method, black reaction in upper layer. Klunge test gave a positive red in the bottom of the tube in a very few minutes.

EXPERIMENT 9.—Miss S. was given 10 grams of chopped beef. The first stool was passed in twenty-nine hours, yellowish-brown, semisolid. Extract, water, guaiac, turpentine method quick change to light green deepening to grass-green with no blue; extract, guaiac, turpentine method, no reaction; extract, guaiac, dilute H_2O_2 method, a brownish reaction in upper layer; extract, guaiac, strong H_2O_2 method, a black reaction in the upper layer. Klunge test gave a reddish-brown reaction intensified by chloroform. Second stool passed on the following day. Extract, water, guaiac, turpentine method, quick change to light green which gradually deepened and faded with no blue; extract, guaiac, dilute H_2O_2 method, no change; extract, guaiac, strong H_2O_2 method, plum-color reaction which did not fade in two hours; extract, guaiac, turpentine method, no change. Klunge test was absolutely negative after half an hour.

EXPERIMENT 10.—Mrs. J. Stomach was washed and thoroughly drained. 10 grams of chopped beef was given at 11.45 A.M. First stool was passed in eighteen hours, yellowish-brown dry cylinders; all tests were negative. Second stool was not saved.

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[8] A grass-green color quickly appears, this intensifies to a deep green, and very gradually fades away.

Conclusions. 1. In order that a positive reaction for occult blood be obtained after eating uncooked meat there must have been ingested 10 grams of beef. The smallest amount to give a distinct blue reaction with the guaiac tests was 20 grams. Of five tests below 20 grams the reaction was positive twice, negative three times.

2. The reaction if positive always occurs in the first stool passed after eating the test meal. This occurred in one case as early as ten hours, in another as late as twenty-nine hours. Only once did it occur with the second stool and never with the third stool.

3. The water modification of the guaiac test gives the most uniform results. Both hydrogen peroxide methods and Klunge's test were at times as sensitive as the water modification. The Boas reaction with turpentine is far less sensitive than any of the others. I have been unable after many trials to obtain a reaction with less than 10 grams of beef. Weber reports positive results with 3 grams of meat.

Series III.—Blood and Stool Mixture Experiments.

The purpose of this series of experiments was to determine the sensitiveness of the guaiac and the aloin tests. A definite amount of blood was added to a definite amount of stool. The quantity in each was determined by exact weight. The first three experiments were conducted by calibrating a capillary tube so that the weight of each drop would correspond. Fearing that this was not as accurate as it should be the remaining tests were made by determining exactly the weight of the blood on a very delicate balance with agate bearings and the exclusion of currents of air. The blood in each experiment was always taken from the same person. It was found that in the cases in which the calibrated tube was employed a drop of blood from one person differed in weight from that of another person, but corresponded quite closely with another drop from the same person. For this reason more than one experiment was necessary in order that definite conclusions could be reached.

Experiment 1.—Drop method. 0.005 gram of blood was added to 1 gram of feces; guaiac, turpentine method was negative; Klunge aloin test was negative. 0.010 gram of blood was added to 1 gram of feces; guaiac, turpentine method was negative; Klunge aloin test was negative. 0.015 gram of blood was added to 1 gram of feces; guaiac, turpentine method was negative; Klunge aloin test was negative. 0.020 gram of blood was added to 1 gram of feces; guaiac, turpentine method gave a distinct greenish color; Klunge aloin test gave a distinct but light cherry color. 0.025 gram of blood was added to 1 gram of stool; guaiac, turpentine method gave a distinct blue color; Klunge aloin test gave a distinct but light cherry color.

Experiment 2.—Drop method. 0.005 gram of blood was added to 1 gram of feces; guaiac, turpentine method gave a light greenish tinge; Klunge aloin test gave a questionable pinkiness. 0.010 gram of blood was added to 1 gram of feces; guaiac, turpentine method gave a light greenish color; Klunge aloin test gave a very slight pinkiness. 0.015 gram of blood was added to 1 gram of feces; guaiac, turpentine method gave a questionable color change; Klunge aloin test was questionable. 0.020 gram of blood was added to 1 gram of feces; guaiac, turpentine method gave a positive blue color; Klunge aloin test gave a positive cherry. 0.025 gram of blood was added to 1 gram of feces; guaiac, turpentine method gave a positive blue to purple; Klunge aloin test gave a very positive cherry.

Experiment 3.—Drop method. 0.005 gram of blood was added to 1 gram of feces; guaiac, turpentine method was negative; Klunge aloin test was negative. 0.010 gram of blood was added to 1 gram of feces; guaiac, turpentine method was negative; Klunge aloin test was negative. 0.015 gram of blood was added to 1 gram of feces; guaiac, turpentine method gave a greenish tinge, positive; aloin test was questionably negative. 0.020 gram of blood was added to 1 gram of feces; guaiac, turpentine method, which gave a distinct pale green, was positive; Klunge aloin test was questionably negative. 0.025 gram of blood was added to 1 gram of feces; guaiac, turpentine method, distinct blue to purple, was positive; Klunge aloin test, distinct but light cherry, was positive.

Experiment 4.—Exact weight method. 0.002 gram of blood was added to 1 gram of feces; guaiac, turpentine method, no change in color, was negative; Klunge aloin test, no change in color, was negative. 0.003 gram of blood was added to 1 gram of feces; guaiac, turpentine method, no change in color, was negative; Klunge aloin test, no change in color, was negative. 0.004 gram of blood was added to 1 gram of feces; guaiac, turpentine method, light greenish tinge, which faded, was positive; Klunge aloin test, upper portion of tube opalescent, was questionable. 0.005 gram of blood was added to 1 gram of feces; guaiac, turpentine method, light greenish tinge, which faded, was positive; Klunge aloin test, upper portion of tube pinkish, was questionable. 0.006 gram of blood was added to 1 gram of feces; guaiac, turpentine method, distinct light green, which faded, was positive; Klunge aloin test was questionable. 0.007 gram of blood was added to 1 gram of feces; guaiac, turpentine method, distinctly deeper green, which faded, was positive; Klunge aloin test, same as before, was questionable. 0.008 gram of blood was added to 1 gram of feces; guaiac, turpentine method, suggestion of blue, was positive; Klunge aloin test, same as before, was questionable. 0.009 gram of blood was added to 1 gram of feces, guaiac, turpentine method, pale smoky blue; which faded, was positive; Klunge aloin test was questionable. 0.010 gram of blood was added to 1 gram of feces; guaiac, turpentine method, pale blue, which faded, was positive; Klunge aloin test, same as before, was questionable. 0.015 gram of blood was added to 1 gram of feces; guaiac, turpentine method, deep purple-blue, which faded, was positive; Klunge aloin test, same as before, was questionable.

Experiment 5.—Exact weight method. 0.002 gram of blood was added to 1 gram of feces; guaiac, turpentine method, no change in color, was negative; Klunge aloin test, no change in color, was negative. 0.005 gram of blood was added to 1 gram of feces; guaiac, turpentine method, light greenish color, which faded, was positive; Klunge aloin test, no change in color, was negative. 0.010 gram of blood was added to 1 gram of feces; guaiac, turpentine method, positive green to blue, which faded, was positive; Klunge aloin test, no change in color, was negative. 0.015 gram of blood was added to 1 gram of feces; guaiac, turpentine method, deep purple blue, which faded slowly, was positive; Klunge aloin test, questionable pinkiness, was questionable.

Experiment 6.—Exact weight method. 0.004 gram of blood was added to 1 gram of feces; guaiac turpentine method, quick change to light green which gradually changed to smoky blue, was positive; guaiac, dilute peroxide method, quick change to light green, which very quickly faded without a suggestion of blue, was positive; water, guaiac, turpentine method, which gave a beautiful light blue, appeared and gradually faded, was positive; Klunge aloin test, no change in color, was negative. 0.003 gram of blood was added to 1 gram of feces; guaiac, turpentine method, absolutely no change in color, was negative; guaiac, dilute peroxide method, very quick change to grass-green, no suggestion of blue, was positive; water, guaiac, turpentine method, immediate change to greenish-blue which gradually changed to a pale blue having a green quality, was positive; Klunge aloin test, no change in color, was negative. 0.002 gram of blood added to 1 gram of feces; guaiac, turpentine method was absolutely negative; guaiac, dilute peroxide method, no change in color, was negative; water, guaiac, turpentine method, which gave a distinct, quickly appearing flat green with a suggestion of blue, which faded, was positive; Klunge aloin test gave no change, was negative. 0.001 gram of blood added to 1 gram of feces; guaiac, turpentine method, which gave a very delicate green color which faded, was positive; guaiac, dilute peroxide method, gave no change in color, was negative; water, turpentine, guaiac method, which gave a beautiful sky blue which faded, was positive; Klunge aloin test, which gave no change in color, was negative. 0.0005 gram of blood added to 1 gram of feces; guaiac, turpentine method, which gave no change, was negative; guaiac, dilute peroxide method, which gave no change, was negative; water, guaiac, turpentine method, which gave a very faint tinge of green but no blue, faded, was positive.

This patient's stools had been tested for several days previous to the experiment, as a control. They had always given a negative reaction with all tests. The stool with which this experiment was made was soft, formed, and of a light-yellow color.

Experiment 7.—Exact weight method. 0.004 gram of blood was added to 1 gram of feces; guaiac, turpentine method, light green changing to smoky blue, was positive; guaiac, dilute peroxide of hydrogen, pale green appeared quickly and soon disappeared, with no blue, was positive; water, guaiac, turpentine method, which gave a beautiful blue, which faded, was positive; Klunge aloin test, which gave no change, was negative. 0.003 gram of blood was added to 1 gram of feces; guaiac, turpentine method, which gave no change of color after repeated tests, was negative; guaiac, dilute hydrogen peroxide, which gave a very quick change to a distinct grass green, with no blue, was positive; water, guaiac, turpentine method,which gave an immediate change to greenish-blue, gradually developing a pale-blue color which faded, was positive; Klunge aloin test, which gave no change in color, was negative. 0.002 gram of blood was added to 1 gram of feces; guaiac, turpentine method which gave no change, was negative; guaiac, hydrogen peroxide method, which gave no change, was negative; water, guaiac, turpentine method, which gave a distinct and quickly appearing cloudy green with a suggestion of blue which faded, was positive; Klunge aloin test, no change, was negative. 0.001 gram of blood was added to 1 gram of feces; guaiac, turpentine method, which gave no change of color, was negative; guaiac, dilute hydrogen peroxide method, which gave no change, was negative; water, guaiac, turpentine method, which gave a beautiful sky blue, which gradually appeared and disappeared, was positive; Klunge aloin test was negative. 0.0005 gram of blood was added to 1 gram of feces; guaiac, turpentine method, was negative; guaiac, dilute peroxide method, was negative; water, guaiac, turpentine method, which gave a beautiful delicate sky blue color, which faded, was positive; Klunge aloin test was negative.

The stool with which all these tests were made was of a light-yellow color and thoroughly soft throughout. It was a semiformed stool and the blood could be triturated into it in a most thorough manner.

Experiment 8.—0.0004 gram of blood, by exact weight, was added to 1 gram of stool and very thoroughly mixed; turpentine, guaiac method (Boas) was absolutely negative; guaiac and dilute hydrogen peroxide method (Boas) was negative; author's water, guaiac, and turpentine method showed a distinct blue ring on the top of the liquid; Klunge aloin reaction was absolutely negative; control was negative.

Experiment 9.—0.00035 gram of blood, by exact weight, was added to 1 gram of feces, and thoroughly mixed; guaiac, turpentine method (Boas) was negative; guaiac, dilute hydrogen peroxide method (Boas) was negative; guaiac, undilute hydrogen peroxide was negative; author's water, guaiac, and turpentine method showed a faint but distinct greenish color in the top of the liquid which quickly appeared and gradually disappeared; Klunge test was negative; control was negative. Below this dilution the water method failed.

Conclusions. From the foregoing experiments the following deductions can be made:

1. The guaiac reaction in all of its modifications is constantly markedly more sensitive than the Klunge aloin reaction. There are times when the aloin reaction seems as sensitive as the guaiac (Experiment 1), but the drop method is not trustworthy.
2. The limit of sensitiveness of the original Boas turpentine method is 0.004 gram of blood per gram of feces. Under certain circumstances it may recognize 0.001 gram of blood per gram of stool (Experiment 6).
3. The limit of sensitiveness of the Boas test when dilute hydrogen peroxide is used is 0.003 gram of blood per gram of stool.
4. The limit of sensitiveness of the author's water and turpentine modification is 0.00035 gram of blood per gram of feces.
5. A color that appears and gradually fades is the crucial test of a positive guaiac reaction.
6. The limit of sensitiveness of the Klunge aloin test is 0.02 gram of blood in 1 gram of feces. Under certain conditions, as in Experiments 4 and 5, a questionable or very faint reaction occurs with 0.015 and 0.004 gram respectively.
7. Most authors agree that it is necessary to obtain a distinct blue color before the reaction can be called positive. Schmilinski,[9] however, is not of this opinion. He regards all shades that develop from a deep green to a reddish brown changing into a violet in which the red is more prominent, and finally the blue predominates as positive as the blue itself. My experience enables me to confirm Schmilinski's contention, as well as to carry it a step farther, and say that all colors that result, from a light distinct tinge of green to green, green to deep green in which at times, but seldom, there is a reddish quality, light violet to blue, or deep blue to indigo blue, are positive evidence that blood is present.
8. The color index may be used as an approximate means of estimating the extent of the hemorrhage. In this series of experiments there was never a change in color with the control, nor was there ever a change in color in the many stools we have examined in the laboratory and marked negative. There was no change,

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[9] Münch. med. Woch., 1903, Nr. 49.

whatsoever, in any experiment until at least 0.00035 gram of blood was added to 1 gram of feces. The first change that was observed was always a light green which became intensified and changed to blue proportionately as the amount of blood per gram was increased. (In one case which showed occult hemorrhages with marked reaction, as the condition subsided the reaction became fainter and fainter until a light green was all that could be obtained, and this eventually disappeared. This is another evidence that the green color is positive of blood.) It may be said that if a light green to a distinct green color develops, a hemorrhage sufficient to mix intimately 0.0005 gram of blood with every gram of feces has occurred; that if a strong reaction is obtained 0.001 gram of blood is present in each gram of feces. As has been shown by the experiments with ingested blood, such a hemorrhage must have amounted to at least 0.5 to 1 gram, provided that it took place in the stomach or jejunum or at some point above these parts.

Series IV.—Blood Ingestion Experiments.

The purpose of this series of experiments was: (1) To determine the amount of human blood that must enter the intestine from the mouth or stomach before a reaction for occult blood will be obtained in the feces. (2) To determine the time that elapses after the ingestion before the reaction is positive. (3) To determine the time that elapses after the appearance of the reaction until it disappears. (4) To determine the comparative sensitiveness of the modifications of the guaiac test and of the aloin test.

To this end blood was taken from the finger of a perfectly healthy person, always from the same person, carefully weighed on a common laboratory balance which weighs quite accurately to tenths of a gram, and quickly mixed with a whisky-glassful of water. The patient was given the contents of the glass to drink, after which it was rinsed and the rinse water swallowed. The stools of these patients were controlled for several days previous to the experiment and were found to be negative. After the ingestion of the blood all bowel movements were saved and the time of their occurrence marked. Sometimes several hours passed before the test was made. It was found, however, that in stools showing reactions for very minute amounts of blood the reaction was not less positive by ageing them for twenty-four hours or more. The results are as follows:

EXPERIMENT 1.—Mr. D. 2 grams of blood was given at 11.45 A.M. First stool passed in fifteen and three-quarter hours; constipated; guaiac, turpentine method (Boas) was negative; guaiac, dilute H_2O_2, negative; guaiac, undilute H_2O_2, negative; author's water, guaiac, and turpentine method, gave a beautiful blue color. Second stool passed in forty-eight hours; guaiac, turpentine method

(Boas) was negative; all other modifications and Klunge aloin test were negative.

Experiment 2.—Mrs. E. 1 gram of blood was given at 11.30 A.M. First stool twenty-one hours; constipated; guaiac turpentine method (Boas) was negative; all other modifications and Klunge aloin test were negative. Second stool passed in forty-six hours; all tests were negative. Third stool passed in seventy-three hours; all tests were negative.

Experiment 3.—Mr. A. 1 gram of blood. First stool, passed in eighteen hours, was formed; author's water method and dilute H_2O_2 method were positive; all other methods were negative. Second stool was passed some hours later, time record lost; all methods were negative.

Experiment 4.—Mrs. C. 0.5 gram of blood was given at 11.30 A.M. First stool was passed in eighteen and one-half hours; slightly constipated; water, guaiac, turpentine method gave a delicate green color, developed slowly, gradually changed to a distinct grass green, and slowly disappeared, was positive; all other methods though repeated several times showed no change of color whatsoever.

Experiment 5.—Mrs. B. 0.5 gram of blood was given at 11.40 A.M. First stool was passed in seventeen and three-quarter hours, hard; guaiac, water, turpentine method gave a pale-green color, but not a typical reaction; all other methods gave no change of color. Second stool was passed in thirty-nine hours; all methods negative.

Experiment 6.—Miss S. 0.5 gram of blood was given at 11.20 A.M. First stool was passed in four and one-quarter hours, mushy; all methods were absolutely negative. Second stool was passed in eighteen and one-half hours, soft; all methods were negative.

Experiment 7.—Miss B. 0.5 gram (fine balance) of blood was given at 3 P.M. First stool was passed in thirteen hours, dark brown and formed; all methods were negative. Second stool was also negative to all tests.

Experiment 8.—Mr. S. 0.5 gram of blood, fine balance, given at 9.25 A.M. First stool was passed in twenty-one hours, brown, and mushy, all methods were negative. The second stool was also negative.

Conclusions. 1. In order that a positive reaction for occult blood occur in the stools after a hemorrhage from the œsophagus or the stomach the hemorrhage must amount to 0.5 gram. With such a hemorrhage only a very faint reaction is present. With a gram of blood a decided reaction is always present.

2. The reaction always occurred in the first stool passed after the blood ingestion. The earliest reaction was obtained fifteen and three-quarter hours after ingestion.

3. The reaction was never present in the second stool passed after ingestion.

4. In this series the water, guaiac, turpentine method was

much more sensitive than any of the other modifications. Next in point of sensitiveness was the guaiac and dilute hydrogen peroxide method. The aloin reaction did not take place in any case.

The foregoing study illustrates the peculiar diversity of results that may be obtained with the different occult blood tests, and at times by the same test, when very minute amounts of blood are dealt with. This not only holds true with feces and blood mixtures but also with stools after the ingestion of blood. In the former case it seems as if the character of the stool has something to do with the sensitiveness of the reaction. A stool with a comparatively large water content, that is, a soft, mushy stool, permits of the recognition of smaller amounts of blood than does a pasty or dry stool. Yet there is a limit to the amount of water that may be added to a pasty stool. In the cases of blood ingestion, it was thought that possibly variations in the sensitiveness of the reaction in different individuals was due to variations in their respective digestive power or to variations in the rapidity with which the food is propelled along the gastrointestinal canal. Thus it was thought that in cases of achlorhydria possibly occult blood would be detected in smaller amounts than in cases of hyperchlorhydria, and that in cases of hypermotility it would be recognized in smaller amounts than in cases showing normal motility or stasis. I soon found that there was nothing in this view, and that after digesting small amounts of blood in test-tubes with stomach fluids of strong digestive power the test was as positive in these tubes as in the control. If, on the other hand, the blood was diluted with water, boiled, and then added to the gastric fluid no occult blood test occurred. The action of the digestive juices has practically nothing to do with the hindrance of the occurrence of the reaction, as has been claimed. The reason for the occurrence of occult blood in cases of achlorhydria is because in this condition erosion is very frequently present.

The character of the stool seemed to have little relation to the sensitiveness of the reaction in the ingestion experiments, as it occurred as readily in a hard dry stool as in a soft one. But these peculiarities are doubtless responsible for the diversity of results obtained by various experimenters. Weber found that after 3 grams of uncooked beef a reaction occurred. I have been unable to verify this contention after many trials, not only with Weber's test, but with other modifications which have always proved more sensitive. Joachim found that 3 grams of ingested blood gave the reaction. With the water modification I was able to get a distinct reaction with 0.5 gram of ingested blood, but was also unable to obtain it with any other test. Schloss obtained a positive reaction when he added 0.0008 to 0.0006 gram of blood to a given amount of stool. Kochmann and Boas obtained a reaction with 0.0008 gram, while

Hartmann obtained it with 0.0001 gram. Schloss is inclined to disbelieve Hartmann's contention and I am unable to demonstrate a reaction with these amounts after many trials. From the diversity of results I have obtained in other experiments, being able to detect with the Boas and Weber tests even smaller amounts of blood than many of these observers have, and still smaller amounts at times with the water modification, I am inclined to believe that the results of all of these men are correct, and that it is possible to detect the minute amount reported by Schloss, if the proper conditions present themselves. In the majority of instances the Boas test with turpentine is made much more sensitive by the addition of water before adding the guaiac resin.

THE RECOGNITION OF MINUTE BLOOD-STAINS ON CLOTHING.

For the recognition of small fresh or dried blood-stains on clothing or other fabrics, I have found the following method to be very sensitive. The test so frequently referred to in works on legal medicine, and often described as Taylor's guaiac method, has not proved as sensitive.

1. Cut out the stain with a pair of scissors.
2. Add from 2 to 10 drops of water according to the size of the stain and rub thoroughly with a glass rod.
3. Add from 2 to 10 drops of glacial acetic acid and mix thoroughly.
4. Add to the resulting solution 1 to 2 c.c. of ether.
5. To the ether extract add a few granules of guaiac resin.
6. Add 20 to 30 drops of old water-white turpentine.

If the stain is due to blood a beautiful blue color develops, continues for about five minutes, sometimes longer, and gradually fades.

Much care must be observed in working with small blood-stains. Too much ether and water dilute the hemoglobin. I have been able to demonstrate a beautiful blue reaction with stains several months old, so small and so thin as to be almost unrecognizable —mere light-brown discolorations. The value of so sensitive a test in legal medicine will be apparent. It does not, of course, differentiate human from lower animal blood. If a stain is large the article tested can then be treated according to the directions given for stool examinations.

General Conclusions. 1. Individuals eating an ordinary meat diet seldom give a reaction in the stools with the occult blood tests. If a reaction occurs it is in all probability due to some pathological process in connection with the gastrointestinal tract.

2. For a positive reaction to occur from the ingestion of uncooked meat, 10 grams must have been eaten; usually 20 grams.

3. For a reaction to occur in the stools after a hemorrhage from

the œsophagus or stomach, the hemorrhage must have amounted to at least 0.5 gram. With such a hemorrhage only a faint reaction is obtained. With 1 gram of blood a decided reaction occurs.

4. The reaction always occurs in the first stool passed after the ingestion of pure blood or of food containing free blood. It seldom occurs in the second stool. It may occur as early as ten, and as late as twenty-one, hours after ingestion.

5. The sensitiveness of the guaiac reaction is greatly enhanced if an equal amount of water is added to the ether extract employed, before the addition of the guaiac and turpentine.

6. The diversity of results obtained by various writers and myself, leads to the conclusion that the results obtained by all can be duplicated if the proper conditions present themselves. In other words, when very minute amounts of blood are present a great variety of results will be obtained by all tests, with the same amount of blood, if experiments are made on successive days.

7. The water modification is uniformly more sensitive than the Weber, Boas, or Klunge tests. All of the guaiac tests are more sensitive than the aloin test.

8. A gradually appearing and fading color is the crucial test of a positive reaction. Any color, from a pale green to deep violet, that develops and fades, is positive.

9. In medicolegal cases, when the question arises as to the nature of minute stains on articles of clothing, paper, etc., the water modification of the guaiac reaction will be found to be more sensitive than the Taylor method, so frequently referred to in works on legal medicine, and its application is very simple.

VARICOSE ANEURYSM OF THE AORTA AND SUPERIOR VENA CAVA.

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On Monday, February 25, 1905, on entering the wards of the Buffalo General Hospital, my attention was at once attracted by the unusual and startling appearance of a patient occupying one of the near beds. He was a large man, in a recumbent position, with a most striking cyanosis of the head, neck, hands, and part of the chest, exposed by his open shirt. He was not blue, but his color was blue-black, of a degree which cannot well be exaggerated. His face was swollen, his eyes closed by œdematous lids. On approaching him I was surprised to find him conscious, and not as distressed and dyspneic as his appearance would lead one to expect. On being addressed, he opened his eyes and gave me a full history in a dull, indifferent, although intelligent manner.

I shall pass over his family history and remote personal history, which throw no light on the condition in which I found him. He told me that for four or five years he had been short of breath on exertion, and had had pain over the precordium. He never had dizziness or œdema of the legs. He said that on the previous Saturday he was at work on Lake Erie, harvesting ice; that in lifting a heavy piece he felt a sudden sharp pain in the right mammary and infraclavicular regions, and felt as if blood or water were rushing up his neck and head on the right side. He became very weak and dizzy, and had to stop work. His neck and face began to swell and felt cold. He had pain in the right chest all the next day, and when he went to bed he noticed that his hands and face were blue. The next day he had mild headache and humming in the ears. He said his vision was somewhat impaired; objects appeared darker than usual. He felt tired and sleepy all day. His bowels did not move and he urinated frequently, small quantities at a time. Later in the day he was admitted to the hospital.

I made a physical examination and quote the following notes made at that time:

A large-framed, well-built, rather plethoric man, aged forty-nine years, with face, head, neck, upper extremities, and thorax extremely cyanosed—almost black—and œdematous. The blue-black color terminates rather abruptly on a line with the diaphragm. His eyes are somewhat bulging—conjunctivæ congested. The veins of the neck, thorax, and abdomen are dilated and tortuous. The blood in the veins flows downward. A heaving impulse is felt in the second, third, and fourth intercostal spaces to the right of the sternum. The sternum is also heaving. The impulse is most marked in the second intercostal space, two-and-one-half inches from the midsternum. The apex is palpable in the sixth interspace one inch outside the nipple line.

The area of cardiac dulness extends one-half inch outside the nipple line. An area of dulness is found in the second, third, and fourth interspaces to the right of the sternum, and two-and-one-quarter inches to the left of the midsternum. There is a systolic murmur at the apex, a diastolic murmur over the aortic area and down the sternum, and a loud systolic souffle over the anterior chest and to the right of the sternum, loudest in second interspace and transmitted to the vessels of the neck and arm and audible also in the abdominal aorta. There is no appreciable tracheal tug. Phonation is harsh. The posterior veins are dilated and tortuous. The percussion note is higher pitched on the right side from above down to the seventh interspace. There is some tenderness in the interscapular area upon deep pressure. A loud systolic souffle is heard posteriorly on both sides, but most distinctly on the right side, and as low down as the crest of the ilium. There is a very slight cyanosis of the lower extremities, due to stasis.

February 28. The condition remains materially unchanged. The patient shows a little more apathy, and quite a little more œdema of the chest and head.

February 29. The patient was presented to the class in the hospital clinic. At this time all of his symptoms were carefully reviewed and verified. No additional notes were made except that there was an absolute want of arterial pulsation in the enlarged and full veins of the neck. Blood examination showed: Hemoglobin 100 per cent. + ; erythrocytes, 6,200,000; leukocytes, 20,000. Urine analysis: slight acidity; albumin, a good trace. Microscopically, there were many small hyaline casts and pus cells. Ophthalmoscopic examination by Dr. Burrows was negative; there was decided increased tension of both eyes (glaucoma).

The signs presented by the patient made it plain that there was an aortic aneurysm, with occlusion of the superior vena cava; but my associate, Dr. Lyon, who saw the patient with me at the time of my first examination, was quick to reach the correct conclusion—that it was a rupture of the aorta into the vena cava. I concurred in the diagnosis, owing to the sudden development of the graver symptoms, which could not be accounted for by aneurysmal dilatation. Reference to the cases reported by Pepper and Griffith,[1] and again by Stengel,[2] made it clear that the case was classic in every detail.

February 30. The œdema increased, breathing became more labored, the hue, if possible, blacker, and the patient died on the fifth day after the onset of his graver symptoms. His temperature ranged between 97.0° and 95°; pulse 120 on admission, gradually declined to 85 before death.

The autopsy revealed the following conditions: Lividity marked; pericardium contained about 200 c.c of serous fluid; the heart, itself, was large and flabby; the mitral valve was normal; the aortic leaflets were thickened; the aorta was calcareous, thick, and on its ascending portion was found an aneurysm extending to the right, and beginning about one inch above the aortic orifice; the sac was about the size of a large fist, and was adherent to the right lung and the superior vena cava. The cava was occluded from a point three-quarters of an inch above the orifice to about two inches above it; the internal walls of the cava were adherent to each other. A small opening about the size of a No. 10 catheter joined the sac and the superior vena cava above the occlusion. The pleural cavity contained serous fluid; the right lung was adherent to the sac and the superior vena cava.

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[1] Trans. Assoc. Amer. Phys., 1890, v, 450.

[2] Ibid., 1900, xv, 532.

THE NATURE OF THE GENERAL TOXIC REACTION FOLLOWING EXPOSURE TO THE X-RAYS.

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In the course of the last few years evidence has been accumulating that *x*-ray exposures may have seriously undesirable effects upon the tissues in the interior of the body, as well as upon the skin and other superficial structures; and of these evil effects that which arouses most marked attention, because it is most dangerous, is the more or less profound and sometimes fatal reaction that has occasionally been observed soon after exposure, and that is characterized chiefly by signs indicating an acute intoxication. It would be superfluous to review the evidence demonstrating the occurrence of such reactions and their definite relation to *x*-ray exposures. Both these points have been shown with sufficient clearness, and both are generally accepted. We have ourselves seen three cases in which a most severe reaction occurred after one exposure to the *x*-rays; two of these cases ended fatally a short time afterward, while the third patient became profoundly ill, but ultimately recovered. In two of these cases the exposure to the *x*-rays was ordered by colleagues; in the other, we were ourselves responsbile for its use.

Since the *x*-rays are of great importance in the diagnosis of many conditions, and apparently also of considerable therapeutic value, it is most earnestly to be desired that the character of the disturbance producing these occasional toxic reactions be determined, in order that we may thereby attempt to devise a well-founded method of preventing the reactions, while still continuing to use the *x*-rays, when they are in place. We have had the rather remarkable fortune of having studied the conditions of metabolism in two such cases, just before, during, and after violent reactions; the cases having been subjected to study for other reasons, and without, of course, any expectation that reactions would occur. We have by this chance been enabled to determine the alterations in metabolism at the time of the reaction, and have obtained results that seem to demonstrate the general nature of the reaction and to sound a warning against the use of the *x*-rays in certain kinds of cases. With us, indeed, these results will constitute a prohibition against the use of *x*-rays in such cases until, at any rate, our knowledge becomes more exact, unless most urgent and exceptional reasons seem to justify us in running some risk in occasional instances.

The cases do not require extended clinical description, and, because both may be reported for other reasons by those who had clinical charge of them, we shall give only the details essential to our purpose.

Case I.—A man, aged forty-three years, with pernicious anemia, was seen in Dr. Stengel's service at the University Hospital. During several weeks of treatment with the usual measures he grew gradually but persistently worse. Owing to the successful results previously observed by Dr. Elliston Morris and one of us in a case of pernicious enemia that was resistant to ordinary treatment, but that improved to a remarkable degree under exposures to the *x*-rays, it was suggested that this agent be tried in this case. At the same time, however, it was urged that the exposure be brief and mild, because in another case of pernicious anemia in which a single exposure had been ordered by Dr. D. J. Milton Miller, which case, as well as that last mentioned, has previously been briefly reported,[1] a violent reaction occurred. It should be especially noted, for reasons that will soon be apparent, that when the exposure occurred, and for some time before, the man presented slight fever, nausea, prostration, and other evidences of a noteworthy degree of intoxication. His exposure was partly over the thighs and partly over the splenic region, its duration was, all told, only five minutes, and its intensity was very mild. Dr. Pancoast, who gave it, informed us that it was as mild an exposure as he had ever administered.

In spite of this caution, about four hours afterward the man became evidently very ill and had a chill. His temperature rose to 103.4°, his pulse became rapid and weak, and he was much prostrated. The fever and prostration continued the next day, and for a week afterward he refused to eat on account of anorexia and more or less nausea. His temperature gradually declined to the previous level and he improved a little, but the gain was, at most, very slight, and while he was still desperately ill his family insisted, about three weeks after the occurrence of the reaction, upon removing him to his home, which was several hours' journey distant, and he died on the train.

The figures for his metabolism follow: It is to be noted that, beginning with the day after the exposure to the *x*-rays, the intake dropped to a very low point. Two days later he refused all food, and the only method of nourishment was through nutritive enemas. We did not determine the amount absorbed, and therefore give no figures for the intake; but from our previous observations on this point and from the fact that he retained his enemas poorly, there is little doubt that the amount of nitrogen absorbed was not above 2.5 grams. The figures given at the end of the table are also not absolutely accurate, as the food at this time could not be so carefully controlled, but they are reasonably accurate and certainly sufficiently generous. The drop in the intake is a point of importance, as will be noted later. The "x" in the second column in the table indicates the date of exposure to the *x*-rays.

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[1] Musser and Edsall. Trans. Assoc. Amer. Phys., 1905; Univ. Penna. Med. Bull., September, 1905.

TABLE I.—URINE.

Date.	*x*-ray exposures.	Nitrogen ingested. Grams.	Nitrogen.	Uric acid.	Phosphates.
Oct. 9	...	10.18	13.892	0.9192	1.170
" 10	...	11.20	14.346	0.9200	1.210
" 11	x	11.83	14.496	0.9594	1.680
" 12	...	9.76	8.400	0.4500	1.164
" 13	...	3.40	10.668	0.4908	2.745
" 14	...	2.50	12.096	0.7630	1.680
" 15	...	...	12.623	0.8376	1.200
" 17	...	...	16.128	0.9292	1.476
" 18	...	...	11.028	0.8769	1.440
" 21	...	...	12.096	0.7406	1.575
" 24	...	2.20	13.608	0.6255	2.131
" 26	...	3.60	11.092	0.8495	1.480
" 31	...	3.00	13.440	0.6836	1.456
Nov. 2	...	2.00	8.400	0.498	1.728

CASE II.—This man, aged fifty-two years, who presented the typical lesions of rheumatoid arthritis, was under the care of Dr. Charles W. Burr, at the Philadelphia Hospital. *x*-ray exposures were ordered by Dr. Burr for the purpose of treating his joint condition; only one such exposure was given, however, owing to the violent illness that followed it. His direct exposure was confined to one arm, from above the elbow down to and including the hand, and was only five minutes in duration; the arm was lying well out from the body, but as the trunk was not screened he got a diffuse and more or less distant exposure of the whole person. It is to be noted in this case, also, that the man was somewhat toxic, having for some time had slight fever, moderate prostration, and occasionally a little delirium at night. These symptoms were apparently the result of a rather severe chronic cystitis. He had no evidence of disease of the kidneys.

After the exposure the temperature rose slightly and he became evidently worse than he had been before. On the second day he was decidedly more prostrated, his temperature was more elevated, and his condition was serious. On the third day he was so much worse that it was thought that he was moribund, and on this day it was impossible to collect his urine accurately, so that the figures for this day are absent. After this he began to mend distinctly, and in a few days had returned to about the condition that he was in before the exposure. The figures for his metabolism follow:

TABLE II.—URINE.

Date.	*x*-ray exposures.	Nitrogen ingested. Grams.	Nitrogen.	Chlorides.	Uric acid.	Phosphates.
Mar. 21	...	12.55	10.1848	6.267	0.1445	1.620
" 22	...	14.10	11.667	6.555	0.3363	1.440
" 23	...	14.45	11.177	4.000	0.3730	1.624
" 24	...	12.74	8.969	3.175	0.4078	1.576
" 25	...	13.34	11.088	6.800	0.4912	1.370
" 26	x	13.22	10.164	6.000	0.4762	1.450
" 27	...	11.85	8.920	4.545	0.4117	1.170
" 28	...	11.96	11.180	8.300	0.6198	1.650
" 29	...	13.24	5.376	4.100	0.2310	0.760
" 30	...	12.26	16.296	7.250	0.7835	2.080
" 31	...	...	Lost	appeared	moribund	
April 1	...	12.15	10.2816	4.330	0.5568	
" 2	...	12.96	7.774	...	0.3318	

The chief point to be noted in regard to the metabolism in these two cases is the remarkable drop in the excretion that followed the exposure, this drop being followed subsequently by an equally striking rise in the excretion to a point much beyond that at which it had previously been. In the first case, the drop occurred directly after the exposure; in the second, it was postponed for two days, but occurred, as in the first case, at the time when the man had become seriously ill. The figures for the intake demonstrate that in neither instance was this drop due to a reduction of the food, for the reduction in the first case occurred after the drop took place, and in the second case there was no marked change in the intake. Likewise, the subsequent rise was not due to the intake, for in the first case the intake was, on the contrary, at this period, very greatly reduced, and in the second case it was not increased. The importance of the great reduction in intake in the first case becomes evident in this connection—the man's excretion from the time of the reaction on must be compared with this reduced intake, and not with the previous intake, in order to appreciate the conditions of metabolism that were actually present at that time.

The only apparent explanation of these changes in metabolism is the following: Previous work has shown that in cases that respond favorably to the action of the *x*-rays an increase in tissue-destruction, that may be of remarkable extent, occurs directly after exposure to the rays. It appears to us that in these cases this occurred: that the organism was overwhelmed by the sudden necessity for carrying on the complete disintegration of a mass of early products of tissue destruction, and that after a little time, as the organism reacted somewhat, complete disintegration could be accomplished and the products were excreted. At first, therefore, owing to sudden severe overtaxing of the organism, metabolism was apparently halted; but the reduction in excretion seems to have been due to mere retention of incompletely decomposed tissue products, and the usual reaction, which is characterized by marked tissue destruction, appears actually to have occurred, though slow in making itself evident, for the later figures show conditions that are quite[2] comparable to those seen in two unresolved pneumonia cases, and in a successful case of leukemia[3] previously reported.

In the first case, in which the reaction was much more severe and certainly hastened death, failure of metabolism occurred at once. In the second case there appears to have been at first some difficulty in getting rid of the tissue products and then an attempt to respond to the action of the *x*-rays, for the excretion rose decidedly on the second day after exposure. But after this a metabolic collapse seems to have occurred, and there was a marked drop in excretion, coincidently with the man's grave symptoms, and this was followed by a very striking output as the condition improved. This latter case is of especial interest in that it perhaps offers an explanation of late toxic reactions; that is, the organism bears up at first under the extra strain, but, sooner or later, collapses as a result of its increased burdens.

We have insisted somewhat upon the fact that both the patients now under discussion appeared to be already the subjects of a noteworthy degree of toxemia. This fact seems to us to help in the explanation of the nature of the reaction and also to offer a probable explanation of the fact that a toxic reaction occurs only occasionally. We believe, from these and other experiences, that a toxic reaction is particularly likely to occur in persons that are already the subjects of a toxemia. Their organisms are already taxed to a considerable degree, and sometimes severely, in attempting to control the intoxication that is already present, and, if to this strain is added the metabolic labor of carrying out the complete decomposition of a large amount of tissue products, it seems quite natural that metabolism should occasionally, in the cases most severely taxed by this, suffer more or less complete collapse; and that, in consequence, the signs of severe intoxication should appear.

This is particularly likely to be true, because the tissue destruction accomplished by the *x*-rays undoubtedly involves chiefly tissues that are especially rich in nucleoprotein. The decomposition products of this form of protein are especially rich in substances that are, of themselves, more or less toxic and also difficult to metabolize and excrete. The figures for the first case, particularly, show that the man soon regained his ability to excrete the main portion of the urinary nitrogen, which comes chiefly from the simpler proteins; and also his ability to excrete phosphates. The uric acid, however, which is the direct indicator of the conditions in regard to the nuclein substances (the products of which are more toxic and more difficult to metabolize and excrete), only slowly regained its previous level. In appreciating the latter point, it should be remembered that the diet was purin-free; and hence that, while changes in the intake influenced the excretion of nitrogen and phosphates, they did not influence the uric-acid excretion.

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[2] Amer. Jour. Med. Sci., 1907, cxxxiii, 286.

[3] Musser and Edsall, loc. cit.

The nature of the toxic reaction to the *x*-rays would then appear to us to be a sudden demand upon the organism for the complete disintegration and excretion of a large amount of the products of tissue breakdown, with inability on the part of the organism to accomplish this, and a consequent halting of metabolism, resulting in an intoxication produced by incompletely disintegrated tissue remnants. That the intoxication in these cases was not dependent directly upon alterations in the excreting power of the kidneys, but upon alterations in metabolism, appears to be at least highly probable, because examination of the urine of these two patients, at the time of the reaction, and for several days afterward, showed no evidence of irritation of the kidneys. It is, however, undoubtedly very probable that in many instances, after a little time, the kidneys do become overtaxed by the added labor thrown upon them, and their excretory power fails to a greater or less degree, and this may increase the toxic symptoms. It is possible, indeed, that moderate but increasing failure of metabolism, together with increasing failure of kidney function, may cause a late accumulation of toxic symptoms and give rise to the late toxic reactions that have been seen both in human cases and in experiments on animals. It seems evident, at any rate, that such symptoms are more likely to occur not only in those that have severe metabolic or infectious intoxications, but likewise in those that have pre-existing kidney disease; and it has, indeed, been already shown in a number of instances that actual nephritis may be set up by exposure to the *x*-rays. Hence, unquestionably, a pre-existing nephritis may readily be made worse by *x*-ray exposures.

If we are correct in all the statements that we have just made, it would appear highly inadvisable, unless urgent reasons to the contrary exist, to use *x*-rays in cases that are already the subjects of a noteworthy degree of general intoxication that is produced by either infectious disease or metabolic disorder. Whether all our observations are correct or not, it would appear that there is at any rate some danger in adding to such an intoxication the sudden necessity for metabolizing and excreting a large amount of the products of tissue breakdown. It has already become evident that cases of nephritis should be exposed to the *x*-rays only with great caution, and our observations emphasize this point still farther.

HYPERTROPHY OF THE ISLANDS OF LANGERHANS IN DIABETES MELLITUS.

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Since the publications of Opie and Ssobolew much attention has been directed to the role of the islands of Langerhans in connection with carbohydrate metabolism and particularly to the changes which are found in them in cases of diabetes mellitus. These changes consist for the most part, as is well known, in degeneration and sclerosis, in which the strands of cells composing the islands are replaced by hyaline-pink staining material. From the frequency of this change and its isolated occurrence in certain cases of diabetes the idea arose that the islands constitute an organ whose duty it is to maintain in some way the ordinary course of carbohydrate metabolism and whose destruction gives rise to such disturbances of this metabolism as are seen in diabetes when sugar is excreted in the urine. Certain observers, however, among whom Hansemann is prominent, think that lesions of the islands are of little importance in the causation of diabetes, and lay more stress upon changes occurring in the pancreatic tissue in general, and Hansemann claims for a type of granular atrophy of the pancreas, which he has described, the chief place in the pathological anatomy of the disease.

There are, however, as Sauerbeck points out in his review of the literature, many cases in which no such definite lesions of the pancreas are to be found and in which the cause of the diabetes must be regarded as extrapancreatic.

Various attempts have been made to give some definite outline to a theory of diabetes without as yet, it is true, arriving at any very lucid working hypothesis. Even if the islands of Langerhans are quite essential to the furtherance of carbohydrate metabolism, as the upholders of that theory believe them to be, we are still at a loss to know the nature of their activities and just where in the chain of events which constitute carbohydrate metabolism their influence is felt. It seemed for a time that with Cohnheim's experiments upon the effect of the interaction of muscle extract and pancreatic extract upon carbohydrates some light had appeared, but more recent work seems to have invalidated to a great extent these results, tempting as they were.

It was with these facts in view that the present case aroused interest, inasmuch as it shows alterations in the islands of Langerhans in the course of diabetes, but alterations which can hardly be interpreted as degenerative changes, nor, indeed, as changes which might bring about their functional insufficiency; on the other hand, they are changes which seem to indicate an actual increase in the bulk and functional capabilities of these islands. The case is as follows:

L. H., a boy, aged ten years, was brought to the Johns Hopkins Hospital on December 14, 1905, complaining of headache, frequent micturition, and extreme thirst. His family and personal history were unimportant. The condition of which he complained had begun three weeks previously with very intense headache. His appetite was not excessive. On entry it was found that he was a somewhat emaciated boy, but physical examination was practically negative. The blood showed approximately normal conditions, except that a little fat was observed in the centrifugalized specimen.

On entry the urine was found to contain 6.6 per cent. of dextrorotatory sugar as estimated by the aid of the polariscope. On December 30, this had decreased to 5.3 per cent. (by Pavy's titration method 5.1 per cent.). Later the amount decreased gradually, being 2.5 per cent. on January 5, and 3.2 per cent. on January 11. No β-oxybutyric acid was present, but acetone and diacetic acid were found in all the more recent examinations.

On February 4 he began to be drowsy and very dull, but physical examination was then quite negative. On February 5 he was still very dull and was given intravenous injections of sodium carbonate. The stupor was such that it was unnecessary to use a local anesthetic in exposing the vein. Very deep respirations with pallor and cyanosis were present at this time. On February 6 air-hunger continued, and, although the patient became brighter after the sodium injection, he lapsed again into coma which did not yield to further injections of alkali. During the injection he suddenly vomited a quantity of fluid, some of which was aspirated into the lungs, with immediate death.

At the autopsy, which was performed a few hours after death, the body was found to be that of an emaciated boy, with dry scaly skin. Examination of the internal organs failed to reveal any gross change whatever, except the presence of food materials in the bronchi. No examination of the central nervous system was permitted.

The pancreas weighed 35 grams. It was normal in color and consistence; on section the ducts appeared normal, and the lobulation and general character of the cut surface could not be said to present any abnormalities.

Microscopically, too, it may be stated briefly, careful examination of the organs, other than the pancreas, failed to reveal any histological abnormality, except for the presence in the liver and renal epithelium of vacuoles containing glycogen.

Chemical examination of portions of the pancreas, which were submitted to Dr. Amberg, showed that the fermentative activities of extracts of the gland were those of a normal gland.

For the rest, portions of the gland were taken from the head, the middle portion, and the tail, and examined in serial section after fixation in Zenker's fluid or in alcohol.

It was found that, in a general way, the pancreas is not much altered, there being no great destruction of parenchyma nor any overgrowth of connective tissue; the lobulation is practically normal, and, for the most part, the acini which make up the lobules cannot be distinguished by any alteration from the normal. The islands of Langerhans, however, which are far more numerous toward the tail than in other portions of the pancreas, are very conspicuous, and seem larger and much more deeply stained than normal. So striking is their appearance that they seem to stand out very prominently throughout the section. Each lobule has such an island in its central portion, and frequently the islands are more numerous and seem to lie close together or even to be almost continuous with one another. Probably these are points in which the somewhat eccentric islands of adjacent lobules approach one another. In size they vary greatly, as seen in serial sections, where the equatorial section may be found, as well as in single sections, where, of course, the smaller areas may always represent merely tangential sections of an island. On the average they are apparently distinctly larger than the normal islands, a basis for which statement may be found in the following table of comparative measurements in which islands were measured in five normal glands:

Autopsy.	No. of islands measured.	Average size.
2685	9	0.132 x 0.156
2662	8	0.104 x 0.100
2679	10	0.200 x 0.168
2693	7	0.172 x 0.128
2698	8	0.180 x 0.180
		average 0.157 x 0.146
2670 (diabetes)	22	0.240 x 0.188

In their histological structure, these islands are quite different from the normal in several respects (Fig. 1). They are far less sharply outlined, having indeed nowhere such a clear contour formed by a membrane or a layer of connective tissue as may often, if not always, seem to encircle the normal island. Instead of this the strands of cells which compose them are almost everywhere distinctly continuous with the cells of the adjacent acini. The transition is perfectly definite and by no means difficult to find or observe (Fig. 2). As one passes from the secreting cells into those of the island, however, a very marked qualitative alteration is observed, as well as the conspicuous change in their arrangement. The cells in the island are quite different in form from those of the acini, being elongated with square ends; they are arranged in long, tortuous, and anastomosing columns, which twist into a sort of knot, and are kept apart by spaces in which lie bloodvessels. Usually only a single row of cells forms these strands, their long axis being

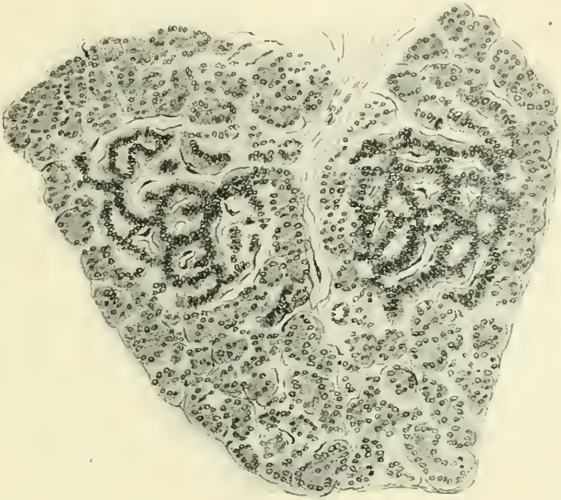

FIG. 1.—Islands of Langerhans of the hypertrophied type, continuous with and probably differentiated from the adjacent parenchyma.

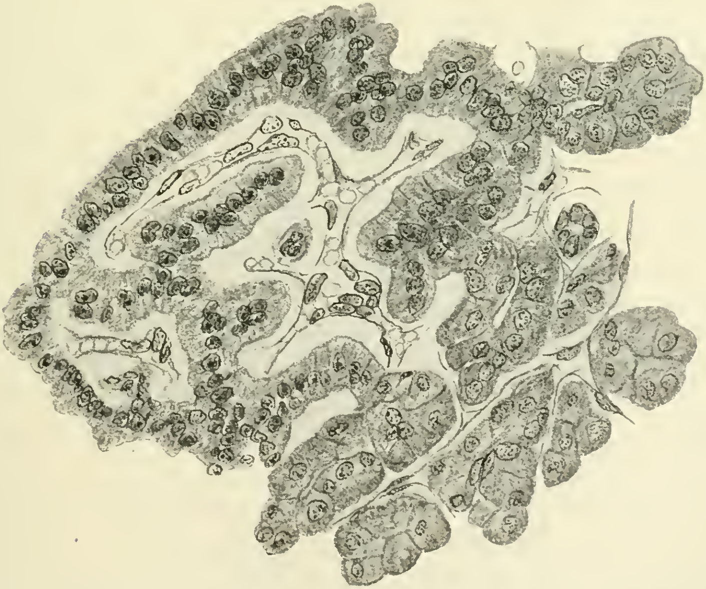

FIG. 2.—Margin of one of the enlarged islands more highly magnified, showing the continuity of the cells of the island with those of the adjacent acini.

arranged transversely to the strand, and their nuclei lying in the middle, so that the whole forms a band of cells with a central row of nuclei. The protoplasm of these cells is less granular than that of the parenchyma in general. The nuclei are large and vesicular and contain coarse chromatin particles, which stain deeply. A large, deeply-stained nucleolus is especially conspicuous, and renders it easy to distinguish these cells from those of the acini.

Such islands, therefore, do not resemble with any precision the normal island of Langerhans. They are not sharply outlined; their columns of cells are long and tortuous and independent, being seldom more than one layer of cells wide; they are everywhere directly continuous at their ends with the adjacent parenchyma; and they stand out prominently by reason of the abundant chromatin content and conspicuous nucleoli of their nuclei.

In many places such strands of cells are intercalated in an isolated way in the general parenchyma, where they are easily recognized; but it is probable, if not certain, that these are always outposts of an island approached tangentially by the knife. They are not found in any connection with the ducts, but lie, as a rule, toward the centre of the lobule. From this mode of occurrence and their general appearance there seems little doubt that they represent the islands of Langerhans.

With few exceptions all the structures found throughout the pancreas, which could be regarded as islands, have these characters. It must be stated, of course, that toward the head of the pancreas there seem to be many lobules without any such bodies. There are also in that part of the pancreas, lobules, in the central portion of which there are no well-defined islands, but in which the parenchyma gives place throughout a relatively small area to a compactly arranged mass of cells, exactly like those forming the islands, but no longer arranged in single rows. In places these cells, less regular and columnar in form than in the above-mentioned islands, are closely applied to one another in such a way that they form irregular radiating masses in which any capillaries present are separated by the thickness of several rows of cells. Such masses resemble only faintly the normal islands of Langerhans. They have neither their arrangement nor their characteristic cells, but are simply radiate masses directly continuous with the adjacent acinar cells, or cells distinguishable by the character of their protoplasm and their deeply stained nuclei.

But there are other lobules which have a quite different look. Chiefly in the tail of the pancreas there are large lobules, few in number and collected closely together, in which the islands of Langerhans are much more like the normal and do not exceed the normal in size. These are sharply and smoothly outlined by a thread of fibrous tissue (Figs. 3 and 4), which separates them clearly from the surrounding acini, at least at most points. In their structure and in the appearance of these cells, on closer inspection, however, they too are very different from the normal; they are made up of rather large columns of cells separated from one another, not by spaces, but merely by the capillaries to whose walls they are closely

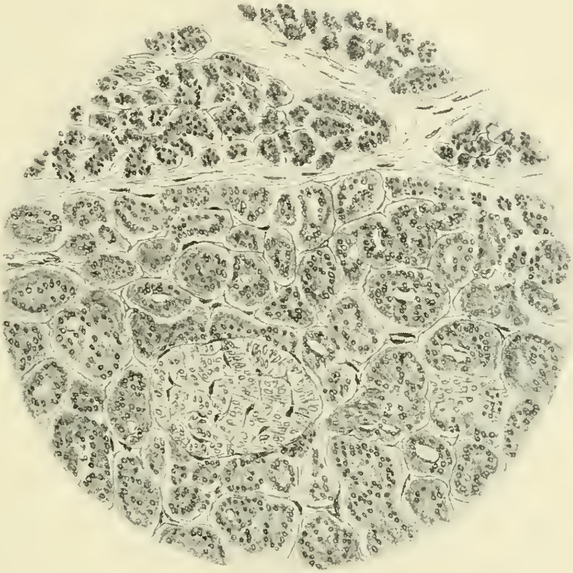

FIG. 3.—Portion of an enlarged lobule, showing enlarged acini and an island of Langerhans more closely approaching the normal. In the adjacent lobule the parenchyma is normal.

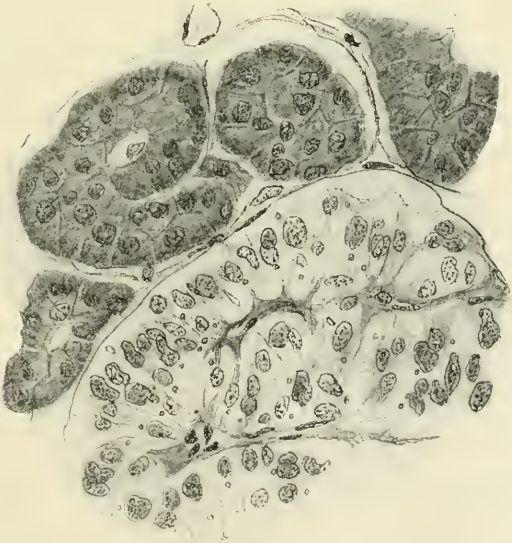

FIG. 4.—Margin of the island shown in Fig. 3. The adjacent acini of the parenchyma are larger than normal. The drawing shows the character of the cells of the island with the peculiar small bodies at the nuclear pole.

applied. Were it not for the great transparency of the cells themselves, such islands would appear very compact, but the cells seem to consist of only the merest ragged outline of protoplasm. The nuclei stain rather palely and do not present so distinct a nucleolus as in the cells of the other islands. They are often accompanied by peculiar small, rounded, bodies which lie near their poles and which take a faint nuclear stain. The nature of these small bodies is not clear.

The acinar tissue which makes up the lobules in which this type of island lies, is different from that in the other lobules, in that the acini and their component cells are much larger. This may be seen in Figs. 3 and 4; but it is much more striking when one can compare under a low power the appearance of such lobules with that of the adjacent ones. That there is an actual increase in the bulk of the tissue seems evident from the fact that sometimes not all of the tissue of the lobule has undergone this enlargement, but only portions of it which then stand out as overgrown masses which push aside and flatten out into concentric rings the adjacent small acini.

Otherwise, however, I can discern no especial change in the character of the cells and no mitotic figures were found.

Another case of a similar character, to which my attention was called by Dr. Opie, occurred also at this Hospital. It was as follows: A child was admitted to the hospital complaining of hunger and thirst, frequent micturition, and excessive loss in weight. The intense symptoms developed suddenly three weeks before entrance. On admission the child was found much emaciated; the skin extremely dry and harsh. Physical examination was negative. The child was semiconscious, and the respiration was labored. The urine contained 2 per cent. of sugar. Acetone was present, as shown by Le Noble's test. There was also a trace of albumin. The patient died in coma, two days after admission.

The anatomical diagnosis reads as follows: Diabetes mellitus; subserous hemorrhages; hemorrhagic gastroenteritis with ulceration; hemorrhage into the intestine; extreme fatty degeneration of the organs. The organs were practically normal throughout, except for the ulcerative condition of the mucosa of the intestine and for the pallor and accumulations of fat in the kidneys and liver. The blood showed a little free fat. The central nervous system was not examined. The pancreas weighed 25 grams, was soft, and perhaps more opaque looking than normal.

In this case the pancreas presents an appearance almost precisely similar to that described above. Only two sections are at my disposal, but from these it appears that there is not only a change in the islands resembling that first mentioned, but also an increase in the bulk of the secreting tissue in certain lobules. The islands, however, in this case, are fairly sharply outlined as in the normal and are all of one kind. The swollen parenchyma shows very distinctly a basal layer of darkly stained granular protoplasm, as contrasted with the paler more central part.

On the whole, therefore, we have in these cases of diabetes in children a peculiar condition of the pancreas, in which the islands of Langerhans are apparently increased in size and number, and modified in their general appearance, and in which certain portions of the parenchyma are also increased in bulk. It remains for us to explain the relation of these changes to the diabetes.

This is a difficult problem in view of our ignorance of the nature of diabetes; but even a working hypothesis may be of some value, and it has occurred to me that there is here the result of some disturbance in carbohydrate metabolism from extrapancreatic causes such as impose an excessive amount of work upon the islands of Langerhans, and that these in order that they may maintain their control of their own particular link in the process have undergone the compensatory hypertrophy which has been described. That this explanation of hypertrophy in an organ of internal secretion is not entirely fanciful is shown by the condition of the parathyroids in a case of gastric tetany, in an old man, in which the parathyroids were greatly hypertrophied in the attempt to maintain their function of neutralizing poisons produced in the stomach.[1] In that case, however, the accumulation of poisonous materials in the highly dilated stomach was such that despite the great size of the parathyroids the man died of tetany. So here in spite of the great increase in the bulk of tissue of the islands of Langerhans the children died of diabetes.

It is also possible, and perhaps even probable, that these islands of Langerhans are not only hypertrophied, but indeed actually newly formed from the tissue of the pancreas. It may be that they have appeared in the tissue of the child's pancreas by a differentiation of Langerhans tissue from the ordinary parenchyma which might not be possible at a more advanced age. This view is supported by the very intimate connection which amounts to actual continuity with the cells of the acini. In that case the presence of a few islands of Langerhans much more closely approaching the normal would suggest that they were the only remaining islands of those originally present, and that the others had been destroyed by some specific process, constituting in that case an intra-pancreatic cause of the diabetes. Thus, two rather distinct hypotheses are suggested: the first, that the excess of Langerhans tissue is the response to an extrapancreatic cause of diabetes, and the second that the destruction and disappearance of all but a few of the islands of Langerhans produced diabetes, but also subsequently a regeneration of islands from the parenchyma. There are no remains or scars, it is true, in the position of the destroyed islands and those now in existence occupy their central position fairly accurately, but still the possibility of this mode of genesis of the condition cannot be ruled out; and, indeed, it might explain very well the increase in bulk of some of the secreting parenchyma which could result from the coincident destruction of similar tissue elsewhere in the pancreas. That the islands of Langerhans are now in a degenerated condition and incapable of function might perhaps be suggested as the cause of the diabetes from their altered appearance, but the idea can hardly be entertained, since their cells are not degenerated and have every appearance of functioning actively. Certainly, however, whatever the mechanism of its immediate production, there is an increased bulk of Langerhans tissue with a coincident diabetes, and whether the cause of the diabetes be intrapancreatic or extrapancreatic, it seems clear that the hypertrophy of the Langerhans tissue is compensatory in nature, as far as it goes, even though it were finally inadequate to avert death.

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[1] MacCallum. Die Beziehung der Parathyroiddrüsen zu Tetanie. Centralbl. f. allg. Path., 1905, xvi, 385.

But little in the literature seems to bear directly on the point discussed here. M. B. Schmidt,[2] in describing extensively altered glands in diabetes, found island-like structures similar to those first described, and regarded them as the result of the differentiation of island tissue from the parenchyma. Gutmann,[3] in one case of pernicious anemia and in one case of diabetes, found somewhat similar newly formed islands, which he also regards as being developed from the parenchyma by a process of differentiation. For this process he finds, however, no adequate explanation, since there seems to be no destruction of the pancreas sufficient to call forth such a response.

UNIVERSAL ITCHING WITHOUT SKIN LESION; HEMATOGENOUS UROBILINURIA; MALARIAL POISONING; PECULIAR ERYTHROCYTOLYSIS.

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The case here detailed is believed to be not only unusual but to present a condition not hitherto described, so far as the relation of the cause and its effects are concerned. The innumerable possibilities of diagnosis were gradually reduced as the study of the symptoms and excretory changes proceeded, until it seems to us certain that but one theory can be held about the origin of the blood disorder and its accompanying manifestations.

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[2] Münch. med. Woch., 1901, No. 41.

[3] Virchow's Archiv, vol. clxxvii, Supplementheft.

While the high eosinophile count which was one of the peculiar features of the case remains unaccounted for except as a part of a curious and unusual blood condition, we believe that all the hemic and urinary changes, as well as the distressing symptoms of Raynaud's disease and of the persistent itching, are accounted for by the destructive erythrocytolysis set up by twenty-five years of chronic malarial poisoning. Although direct evidence of this malarial poisoning cannot at present be found, we consider it sufficiently proved not only from the blood and other signs, but by the exclusion of other possible causes.

The patient, M. H., aged fifty years, was born in Pennsylvania of German-Hebrew parentage. The family history is good; his father died at seventy-two of "heart failure;" his mother is living, aged eighty-five; a brother and four sisters are alive; the only doubtful point is that a sister is said to have died of "anemia" at twenty-three years old. She had been ill for four or five years with some nervous disturbance following typhoid fever, but was married, became pregnant, miscarried at two months, it is said from "weakness," and died a few months later of a "decline," probably, but not certainly, phthisical. Mr. H. is married and has one healthy child. He has had no illnesses except the diseases of childhood, but has always been nervous and easily perturbed, with a tendency to depression. His weight is 135 pounds, usually 150 pounds; his height, 5 feet, 7 inches.

He has lived in California in various places from 1859 to the present, except from 1865–1870. From 1876 to 1902 he was in a region, Willows, described as "malarial." Many persons suffered there with obscure forms of illness, supposed malarial, though frank chills and fever were uncommon. During his residence in this district he had various discomforts and ailments like those of masked or undeclared malaria, such as leg-ache, head-ache, nape-ache, and biliousness, for seven or eight years. The fact that quinine relieved all the symptoms in him and in the other residents of the district makes it sufficiently certain that they were of malarial origin.

His habits have always been sedentary; there are no excesses, venereal, alcoholic or other, admitted. He smoked for many years largely, but never, so far as known, with deleterious results and not to an extravagant extent, six or seven light cigars daily.

A mild form of Raynaud's disease has been present for seven or eight years, beginning soon after the malarial infection described. His hands become mottled blue and white and swell greatly in cold weather, and are pink in warm weather. They perspire very little and are subjectively cold.

His present complaint is stated to have begun in 1902, or even a little earlier, with constant indigestion, severe epigastric distress, lasting five or six months. During this time he first suffered from the main symptom which has distressed him ever since, namely, an intolerable itching of the whole body. No lesion of the skin has ever been present, no discoloration, no jaundice, no hepatic colic. The itching grew better for a time when the digestive disturbance was treated by lavage, but recurred afterward worse than ever with no return of the digestive symptoms. In January, 1903, he broke down nervously, partly from the loss of sleep caused by the itching. He made some improvement, but relapsed, and in October, 1904, was ordered a sort of rest treatment, very incomplete in character. There was again some betterment, but the itching returned, his sleep became more broken, and a condition of considerable mental depression followed.

Various treatments, local and general, varying from applications of carbolic acid, menthol, and olive oil to warm baths, at various times brought some relief but no permanent change for the better.

When seen in April, 1905, Mr. H.'s general appearance was fairly good. His color was not deficient, and though his skin was not clear and had a slightly muddy look, there was no icterus. His complaint was of general nervousness, inability to apply himself to work, some depression, constant general itching, much worse at night, with broken sleep.

His station was slightly unsteady; knee-jerks, weak, small, reinforcible; pupils reacting normally; other reflexes normal; no pain or girdle sensation; bowels normal; lungs clear; heart rhythmic, doubtful mitral systolic murmur, second sound clear; compensation perfect. The patient was not aware of any cardiac trouble and had no symptoms of deficient cardiac function. Pulse tension (radial) good.

The blood and urine were examined with care on April 19, and as the diagnostic interest of the disease centres in the blood and urinary findings, these must be given in detail. Before the minute study of the urine could be made the peculiar port-wine hue which it assumed on standing attracted attention and suggested a search for various coloring materials, melanin, melanogen, phenol, bilirubin, urobilin, blood, and bile acids. Numerous tests for all these proved negative except for urobilin, which was positively demonstrated as present by several chemical methods, and most definitely by its peculiar spectroscopic absorption band. We consider that this urobilin was not derived through bilirubin, but directly from hemoglobin, for the following reasons:

(*a*) No bile acids or bile pigments were present at any time.

(*b*) The liver functions were unimpaired so far as the nitrogenous output was concerned.

(*c*) There was no jaundice.

(*d*) Urobilinuria if hepatic in origin is almost always accompanied by an increase of indican, which in this case was never found in a quantity beyond a faint trace, certainly not more than would normally be present.

(*e*) Raynaud's disease was present and with it an hemolysis which although not altogether of the kind often found with Raynaud's might be related to it and be a source of the urobilin.

(*f*) A constant relation was found to exist in our later observations between the degree of hemolysis and the urobilin content, the one increasing or diminishing with the other's changes.[1]

The quantitative urinalysis made on the same date as the study described above was as follows: Twenty-four hours' amount 1240 c.c.; acid reaction; specific gravity, 1021; urea, 26.66; albumin, a trace; sugar, absent; chlorides (*as* NaCl), 8.99; uric acid, 0.804.

Microscopic examination, showed many polymorphonuclear leukocytes, a moderate amount of squamous epithelium, which was distinctly stained yellow, and a few hyaline and pale granular casts.

All the evidence, both positive and negative from the urine analysis, seemed thus in favor of the derivation of the urobilin from the blood rather than from the liver. This view was strengthened by the blood examination, which was as follows: Hemoglobin, 58 per cent.; leukocytes, 19,000; lymphocytes, 35 per cent; large mononuclears, 4 per cent.; transitionals, 4 per cent.; polymorphonuclears, 42 per cent.; eosinophiles, 13 per cent.

There was a very pronounced degeneration of the red blood cells, which showed a number of forms within the erythrocytes. These appeared as non-staining, slightly granular bodies, usually eccentrically located. Sometimes they seemed to be half within and half without the corpuscles and sometimes altogether without and deeply indenting the sides of the erythrocytes against which they lay. Though not very numerous they were well distributed in the slides and in repeated examinations were always present. Efforts to stain them wholly failed.

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[1] In testing for bilirubin the following methods were used: Smith's (W. G. Smith, Dublin Med. Jour., 1876, p. 449), Huppert's (Arch. d. Heilk., 1867, vol. viii, pp. 351 and 476), Gmelin-Rosenbach (Centralbl. f. d. med. Wiss., 1876, vol. xiv, p. 5), Gmelin (Die Verdawung nach Versuchn, Heidelberg, 1826, i, p. 80). In the search for biliary acids the urine was treated as Tyson suggests (Phila. Med. Times, July, 1873): 250 c.c. was evaporated slowly to dryness over a water-bath. The residue well mixed in a glass mortar with an excess of absolute alcohol, filtered and the filtrate treated with from twelve to twenty-four times its volume of ether. In this manner the bile acids, if present, are precipitated. Filter and dissolve the residue in distilled water. Decolorize by passing through animal charcoal. The resulting clear and colorless liquid is subject to Pettenkofer's test.

When the amyl-alcohol extract of the urine was treated with a concentrated alcoholic ammoniacal solution of zinc chloride the resulting mixture showed a strong green fluorescence and further spectroscopic examination gave a marked absorption band of urobilin Indeed, it was found that the urine itself without any treatment gave this band. No effort was made to determine the urobilin quantitatively, as the only practical method is that of Hoppe-Seyler (Virchow's Archiv, 124), and this we consider inaccurate in view of the fact that some urobilin may always be lost in the washing of the chloroform extract, no matter how slightly this be done. The Hoppe-Seyler method was used to isolate the body in its pure state.

The extraordinary eosinophile count, nearly 10 per cent. above the normal, which this examination disclosed, continued high during the whole course of our observation of the case, though later it fell somewhat.

In wet preparations the cells exhibited an excessive tendency to rouleaux formation.

The general type of this degenerative relation was like Maragliano's endoglobular change, as was agreed by Professors Allen Smith and Alfred Stengel and Dr. John C. Da Costa, Jr., all of whom examined with interest many slides of the patient's blood at various times.

No other changes than these were found in the blood in the many examinations made afterward and these persisted in various degrees during the ten months of observation. Special pains were taken to search repeatedly for any form of Plasmodium malariæ, but none was ever found. Repeated attempts were made with various stains upon the granular bodies. With Wright's stain the extruded granular mass took on a deep blue, but they would not color at all with eosinate of methylene blue or with hematoxylin-eosin.

The number of white cells (19,000), the high proportion of eosinophile corpuscles and the presence of transitional forms suggested leukemic conditions, but no general symptoms were present that would point to such a conclusion, and besides the total number of leukocytes diminished very rapidly after treatment was begun (April, 19,000; June, 13,250; October, 5540), although there was no alteration in the eosinophile percentage for some time.

Ehrlich's original statement in describing the affinity of leukocytes for eosin was that this affinity existed only in leukemia, but further observations have shown that this is not true and that eosinophile corpuscles are found in a bewildering variety of diseased states. The possibility of intestinal parasitic infection, a condition which might produce some of the signs and symptoms present, was considered, and careful and often repeated microscopic examinations of the stools after sedimentation were made without discovering any ova or other evidence of the presence of parasites.

Finally, after discussing many theories to account for the eosinophilia and making search without success for any case resembling this one, we put aside the question of the origin of the high percentage of eosinophile corpuscles, believing this to be rather a blood condition related to the general hemic changes than one of special diagnostic significance in the present instance.

The question of treatment had thus to be settled without any very precise basis of diagnosis to rest upon or, at least, without being founded upon any named known and defined disease,

Symptomatically, we had to deal with the slight renal irritation shown by the presence of a few casts and a trace of albumin, with the moderate general anemia, shown by the considerable deficiency in hemoglobin, the large leukocyte count, and, more significantly, by the erythrocytolysis which was present in so marked and peculiar a form. From the patient's point of view the symptoms of general nervousness, insomnia, depression, and above all the nearly constant and very distressing itching to which the insomnia and nervousness seemed largely due—were the important matters. That the itching was caused by the urobilin seemed a reasonable conclusion and that the urobilin had its origin in the breaking down of the red cells appeared equally certain. The pedigree of urobilin, as was said above, is uncertain, that is, it may be derived from either bile pigments or blood pigments. In the present case the bile pigments could be excluded, leaving only the hemic origin probable. Finally, there were the Raynaud symptoms, also thought to be due to the blood disturbance, but more fully to be discussed later.

If, therefore, the blood condition could be improved and the erythrocyte destruction stopped the most annoying symptom would be removed.

Attacking the disorder from this point of view, the patient was ordered tincture of the chloride of iron to be taken in increasing doses for its double effect as a hemic tonic and on kidney irritation, an alkaline laxative (sulphate of magnesium), daily massage, and warm baths containing carbonate of sodium one night and starch solution the next. For the first few nights, $\frac{1}{400}$ grain of atropine sulphate seemed to relieve the itching but soon lost its effect. Mr. H. had had every variety of local application that could be imagined for pruritus, without effect—so no such drugs as menthol, carbolic acid, and the like were tried. After a week or two, small doses of Fowler's solution were ordered and given for about three weeks out of four during the whole of the treatment.

The improvement was marked in the first few weeks, the itching and consequent sleeplessness and nervousness grew less constant and less severe.

In June after about five weeks' treatment the blood count was as follows: Hemoglobin, 72 per cent. (April, 58 per cent.); white cells, 13,000 (April, 19,000); red cells, 5,450,000.

The last week in June, with a small increase in hemoglobin (74 per cent.), other blood conditions remained about the same, the differential count showing the same eosinophile percentage (13), and the same high general proportion of leukocytes. The degenerative changes, however, under the microscope were much less both in extent and in severity. The spectroscopic absorption band of urobilin in the urine was present, but proportionately less marked and the patient was much more comfortable, having very little itching by day and much less than formerly at night.

During the summer a course of alterative medication seemed to do a great deal of good, all the symptoms steadily, though slowly, lessening under a combination of iodide of potassium, bichloride of mercury, and Fowler's solution. Albumin entirely disappeared from the urine and with it the casts went.

Without going needlessly into detail it may be said that the patient continued to progress satisfactorily under the above *regime*, and in November was able to say that he had not in years been so free from itching.

In October, examination of the urine showed it to be practically unchanged, except that there was only a trace of albumin, no casts could be found, and the spectroscopic band of urobilin was fainter than in the summer.

In November no albumin could be demonstrated by any test.

Blood Examination, November 23, 1905. Erythrocytes, 4,607,000; leukocytes, 9111 (April 19,000); hemoglobin, 76 per cent. (April, 58 per cent.); color index, 0.82.

Differential Leukocyte Count. Lymphocytes, 20 per cent.; (April, 35 per cent.); transitional and large mononuclear leukocytes, 17 per cent. (April, 8 per cent.); polymorphonuclear leukocytes, 49 per cent. (April, 42 per cent.); eosinophiles, 13 per cent. (April, 13 per cent.); basophiles, 1 per cent.

There was again marked improvement since the examinations in July and October in the degenerated erythrocytes. At this time sweat baths, followed by a warm, gentle douche, were ordered in place of the alkaline and starch baths. The patient continued to improve until he was discharged and sent home in February, 1906.

The last blood examination made before Mr. H. left Philadelphia showed practically normal red and white count, with a color index of 0.94. The differential count of the leukocytes was only remarkable for a continued excess of eosinophile corpuscles, which had, however, diminished to 6.3 per cent., the normal figure being from 3 to 4 per cent. In the stained specimens very few cells could be found showing the endoglobular change which formerly was present in every field. A very faint spectroscopic absorption band of urobilin was still observable on testing the urine.

We hear since this gentleman underwent the nervous shock, exposure, and physical strain of the San Francisco earthquake that some of his symptoms have reappeared, but we have not received any details that could be added to this interesting case-history, except that he had neglected for some time before the earthquake to follow the line of treatment which he had been ordered to continue, so that this may have had something to do with the partial relapse.

The interest of the case lies in what appears to us a new symptom-complex and in the probable malarial origin of the disorders. It is the combination of these symptoms that is the important feature, for none of them are in themselves extraordinary, except the intense persistent general itching. This latter was, like the itching of icterus, universal, but far more severe and annoying than that of jaundice. The peculiar blood changes, although superficially resembling these of malaria, differed in presenting but one form of alteration in the corpuscles and this form resistant to the stains which have affinity for the plasmodia. Nevertheless, we consider this degeneration due to the malarial poisoning and productive of the urobilin in the circulation to which in turn the itching is due. The evidence for chronic malarial infection seems to us sufficient. First, a residence in a known malarial district, together with frequent attacks during that residence with such symptoms as are produced by masked or undeclared malarial infection; second, the therapeutic test of these symptoms, which both in the patient and in other persons with like troubles were relieved by quinine; and third, the presence of Raynaud's complex. In Raynaud's first description of the combination of symptoms since called by his name (1862) the previous occurrence of malarial fever was noted in some cases and again in the second publication (1874). Other observers have found it so frequently precedent that they have considered an almost necessary relation to exist. Petit and Verneuil,[2] for example, cite over twenty cases of Raynaud's disease occurring in subjects who have had, "even some years before, attacks of intermittent fever, more or less severe, and more or less rebellious to treatment. It is in cases of this type that the etiological relation can be and indeed has been most readily recognized." Other authors have noted the same connection, and some have described in not very definite fashion peculiar blood changes, hemoglobinemia, abnormal tendency to rouleaux formation, stained liquor sanguinis. Apart from these a peculiar granular degeneration in the red blood cells has been frequently found in Raynaud's disease, but it is not like that noted in our case.

As a matter of interest, but not with any special relation to the present discussion, as the existence of the Raynaud's disease requires no confirmation, it may be added that local circulatory symptoms such as began after the malarial troubles are peculiarly common in Hebrews. It may be that this is only another instance of the already well-known fact that nervous disorders occur in that race with especial frequency. The patient is of sound German-Jewish stock, not of the Polish or Russian families that fill our clinics with hysterical and neurasthenic cases. Dr. S. Solis Cohen, of Philadelphia, says that during a year in which he was on the lookout for instances of "circulatory ataxia" he saw in his clinic incidentally about sixty cases of Raynaud's disease, all in Jewish patients who had come under treatment for other disorders.

Finally, the points for emphasis and interest in the case may be thus resumed: persistent universal itching without skin lesion, due to the presence of urobilin in the circulation, which in its turn was the result of a peculiar hemolysis, caused by chronic malarial poisoning; Raynaud's disease, probably of the same malarial origin; slight renal irritation; and general nervousness. The therapeutic and diagnostic interests lie in the failure of local measures to relieve the itching, the mistaken views of the origin of this chief symptom as the result instead of as the cause of the neurasthenic state, the supposition that it had a digestive origin, and the final partial success of treatment directed to the improvement of the blood-making functions, together with such alterative measures as sweat baths and stimulating douches, and the moderate long-continued administration of iodides, mercury, and arsenic.

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[2] Rev. de Chir., 1883.

FIBROID TUMORS OF THE VULVA.

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Benign tumors arising from the external genitalia in women are of infrequent occurrence. The number of reported cases is comparatively insignificant, and if the growths reported be restricted to fibromas, the number is surprisingly small. Thus, Burr,[1] in 5000 gynecological cases, tabulated at the Michigan University Hospital, did not find a case of vulvar fibroma. Williams in studying 420 cases of neoplasms of the external genitalia found but seventeen fibromas, or about one in 600 of all the new-growths arising from the female genitalia that he studied.

The reason for this infrequency is difficult to find. The histology of the labia and of the other tissues about the vaginal outlet would seem rather to predispose this region to stromatogenous new-growths.

Loose connective tissue, with abundant vascular supply, and the presence of many mesoblastic elements with much muscular fiber, should be a fertile soil for the production of such growths. If there be added the irritation from clothing and bodily exercise, and the constant risk of a certain amount of trauma, especially in married women, this predisposition would seem to be still more marked.

The life-history of the labial tissue, as Burr says, is in no sense different from that of similar tissues in other parts of the body. The same phenomena of development, maturity, and atrophy exist here as elsewhere, and the same etiological factors should be in operation here as in other localities where fibromas are more commonly found.

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[1] A Case of Fibroma of the Vulva, New York Med. Jour., 1905, lxxxi, 340.

The number of fibromas is further reduced by the fact that many of the older cases were in reality elephantiasis vulvæ, as is pointed out by Veit.[2]

The old view held and strongly advocated by Scanzoni[3] that these growths are due to the organization of unabsorbed blood clot in the labia is unsupported.

Fibroid tumors of the vulva occur usually in parous women, and toward the end of the period of sexual maturity. In Burr's statistics two cases occurred between the ages of thirty and fifty years, three cases between the ages of twenty and thirty years, and one under twenty years. In the 34 cases collected by myself the age limits were as follows: Under twenty years, 3 cases; twenty to thirty years, 5 cases; thirty to forty years, 7 cases; forty to fifty years, 10 cases; and over fifty years, 3 cases. In 6 cases no age was given.

The great majority of the patients were married and had borne children. Of the 18 cases studied by Burr, only 1 was in an unmarried nulliparous woman. Of the cases that I have studied, 12 were in parous and 5 in nulliparous subjects. They are more common in negroes than in white persons. Ramsey says 12 in negroes, to 3 in white women.

The growths vary greatly in morphology. They are mostly small, ranging from walnut to orange size, but they may reach large proportions. Largeau[4] removed a cystic fibromyoma from the left labium majus that weighed 75 pounds, while Whitney[5] reported a case in which a tumor, having probably the same histogenesis as those under discussion, was found to weigh 268 pounds. The vast majority, however, are small.

The location of the growths is variable. A majority seem to spring from the labia majora, with the preference for the left side. The remainder spring, with almost equal frequency, from the labia minora and the tissues immediately adjacent to the clitoris. In Burr's cases, six grew from the right labium majus, eight from the left, while but one grew from the left and the right labium minus respectively. In 16 cases of my own statistics the tumor grew from the right labium majus, in 17 from the left, and in 1 from both.

The tumors may be pedunculated or sessile. In all probability all are sessile during their early development, becoming pedunculated as they increase in weight and as the constant pull of the tumor mass tends to stretch and attenuate the loose connective tissue in which they are embedded. Nine of Burr's and twenty-one of my own cases were pedunculated. The pedicle should be carefully studied, as it may determine absolutely the point of origin of the tumor. In Largeau's case the pedicle of the tumor ran far back into the pelvis, through the inguinal canal, and apparently to the uterus, although the operator did not care to follow it so far.

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[2] Handbuch der Gynecologie, Band iii, 216.

[3] Lehrbuch der Krankheiten der Weiblichen Sexualorgane, p. 582.

[4] Archiv. prov. de chir., 1892, i, 531.

[5] Subperitoneal Pelvic Fibromata, Annals of Surgery, 1905, xli, 823.

The duration and rate of growth of the labial fibromas vary greatly. They usually grow slowly, but may have periods of great activity, especially during pregnancy, and to a less extent at the menstrual period. Scanzoni calls attention to the fact that in several cases observed by him the tumor became very much congested and increased appreciably in size during the catamenia. Penrose[6] also reports a case in which a large pear-shaped, œdematous fibroid, growing from the right labium majus, increased to double its size at the menstrual epoch.

In appearance, then, the growths are sessile or pedunculated masses, arising from the labial tissue. When pedunculated the pedicle is usually of moderate length (4 to 8 cm.), but many cases are recorded in which the tumor hung between the thighs, being dependent from a long tenuous pedicle. The sessile variety may simulate closely cysts of Bartholin's glands, or even labial hernia. This is especially marked when the tumor has undergone cystic or myxomatous degeneration. The skin is usually roughened and corrugated, but rarely adherent. By reason of pressure upon neighboring skin surfaces, superficial ulceration is frequent; this, however, never becomes marked, though epithelioma may supervene.

The symptoms of these tumors are usually those resulting from the mere presence of a mass in so unfavorable a situation—a sense of weight from the tumor, discomfort in walking and sitting, with occasional sharp pain, should the tumor be struck or pressed upon. When the tumor tends to expand laterally, instead of becoming pedunculated, it may press upon and displace the urethra or meatus, or it may grow at the expense of the vagina, preventing coitos, or even becoming a bar to parturition (Zielewicz[7]).

A curious case is reported by Kirchoff:[8] A girl, aged eighteen years, had for some time felt, when lifting any weight, as though something were trying to force itself out of the vagina. One day while lifting a kettle she experienced a sudden sharp pain, with some hemorrhage from the vagina. Upon examination, a pedunculated fibroid was found, arising from the inner side of the right labium majus, and reaching to the middle of the thigh, while the hymen showed that it had recently been torn by the extrusion of the tumor from the vagina.

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[6] Amer. Jour. Obstet., 1896, xxxiv, 72.

[7] Archiv. f. klin. Chir., 1889, xxxviii, 340.

[8] Cent. f. Gyn. 1893, xvii, 1037.

The diagnosis of these growths is ordinarily not difficult. The presence of a slowly growing sessile or pedunculated tumor in the labium, with the consistency of a fibroid, no skin adhesions, and no evidence of cachexia, should make the diagnosis plain. Hernia into the labium majus, whether intestinal or ovarian, may be a source of error. Tuttle[9] describes a case in which incision into the labium for fibroid revealed a structure resembling a hernial sac. Only after careful examination was it found to be a cystic fibroma, having its origin in the tissue of the round ligament. Fromme[10] also reports a case in which the differential diagnosis between labial hernia, cyst of Bartholin's gland, and solid tumor was uncertain. The growth proved to be a myoxmatous fibroma. The diagnosis in this case was confusing by reason of the patient being a virgin, of eighteen years.

A point of distinction has been made between benign and malignant growths of the vulva, with respect to their location. The benign growths are most frequently found arising from the labia majora, while the malignant neoplasms have their inception in the tissues about the clitoris or the labia minora.

The treatment should always be excision of the growth. The dangers of malignant degeneration are too real to permit of temporizing. Case after case is reported in which sarcomatous degeneration of a fibroid, with its attendant terrors, took place. The discomfort from the presence of the tumor and the comparatively trivial operation required for its extirpation usually render surgical interference a matter of course. The operation is usually simple, but severe hemorrhage may occur.

In Eichholz's[11] case a cavernous fibroid was removed, in which some of the vessels of the pedicle were 12 mm. in diameter. Storer[12] reports a case in which the pedicle was found to consist of a maze of veins and arteries, some of the latter of immense size, one fully as large as the femoral.

The case coming under my notice is as follows: M. S., American, married, forty-five years, VI-para, was admitted to the Gynecean Hospital October 31, 1905. She had always been a healthy woman, with no serious illness nor hereditary stigmata. There was no history and no evidence of any specific infection. Her marital life had been without incident.

Physical examination revealed no organic disease, Eight years before admission she noticed a small hard lump in the left labium

[9] Amer. Jour. Obst., 1891, xxiv, 114.

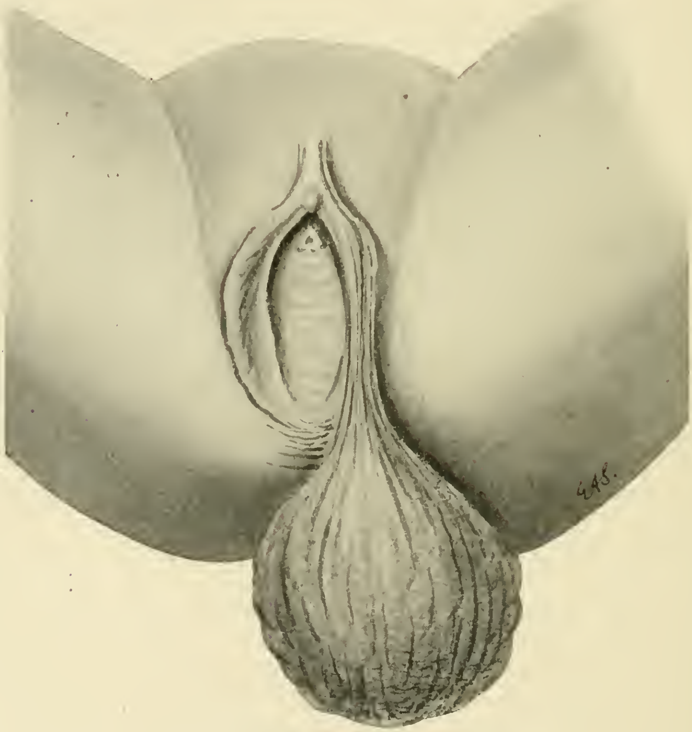
[10] Monatssch. f. Geb. u. Gyn., 1904, xx, 961.

[11] Frauenarzt, 1892, vii, 520.

[12] Gynecological Journal, 1871, ix, 326.

majus. This increased in size steadily but slowly, until, at the time of examination, it hung as a mass, the size and shape of a pear, swinging between the thighs, and attached to the much thinned-out left labium majus by a pedicle, about 2 cm. in diameter and 10 cm. in length (see figure). Upon the inner surface of the tumor there was a circular, sharply defined, sloughing ulcer, 15 mm. in diameter.

Under ether anesthesia the tumor was extirpated without difficulty. The hemorrhage was slight and easily controlled. The wound was closed by buried and superficial catgut sutures. The convalescence was uneventful.



Myxomatous fibroma of the labium majus.

The specimen consists of a tumor dependent from a pedicle 10 cm. in length, which had spread out the left labium majus. The growth measures about 6 cm. in diameter, is semi-fluctuating, entirely covered by skin. Where it came in contact with the skin of the thighs there is a punched-out ulcer, 15 mm. in diameter. The ulcer is shallow and its base is occupied by a grayish slough.

Microscopically, the neoplasm is a fibroma undergoing myxomatous degeneration. The entire picture is hazy; the nuclei of the cells stain slightly and show characteristic irregular prolongations. No granules of precipitated mucin can be discovered.

The chief interest in fibromas of the labia centers about their histogenesis. The simplest explanation is that they develop from the subcutaneous connective tissue, in the mesodermic tissues, so abundant in this region, or along the adventitia of the bloodvessels, as do most uterine fibroids. This manner of development has been strongly upheld by Klob[13] and others.

But it has undoubtedly been shown that many of these growths originate in much deeper structures. Fromme described a case in which there was found a fist-sized, fluctuating tumor, occupying the entire right side of the vulva, forcing the vaginal outlet to the left. The tumor was immediately under the skin. After removal the growth was found to be uniformly solid in texture, there being no cysts. Under the microscope the tumor was found to consist of a connective tissue capsule, enclosing the tumor itself, which was very rich in connective tissue, with a few muscle fibers. Throughout the entire growth there were noticed single, tubular glands, lined with a single layer of cylindrical epithelium. These glands gave evidence of being offshoots from Bartholin's gland. The writer concludes that the tumor must have sprung from the interstitial connective tissue in Bartholin's gland, and in evidence cites a case of von Recklinghausen in which there existed hypertrophy of the glandular connective tissue of Bartholin's gland. This establishes a second source of such neoplasms.

Kewisch[14] maintains that the tumors spring from the periosteum of the pelvic bones, or from the deep pelvic fascia, and supports the argument with illustrative cases. It may be, as Storer[15] has pointed out, that vulvar fibromas may arise from the connective tissues of the rectovaginal septum, and reach the labia by following lines of least resistance. Sanger[16] outlines another point of origin of labial tumors: the extraperitoneal portion of the round ligament, and he classifies these growths into four groups: (1) Intraperitoneal—the growth extending along the course of the round ligament from the uterine cornea to the internal inguinal ring. (2) Intracanilicular—the growth being entirely within the inguinal canal. (3) Extraperitoneal—the growth being in the abdominal wall, outside the inguinal canal, or in the labia majora. This group is subdivided into the tumors with pedicles and those which remain sessile. (4) Growths occurring in remote regions of the abdominal wall, but having a histological connection with the round ligament.

It is the third, or extraperitoneal group of tumors, which particularly concerns us. A few typically illustrative cases of this type have been reported. Nebesky[17] describes such a tumor, of apple size, arising from the left labium majus, and of slow growth. Upon removal of the tumor, there was found a fibromuscular cord leading up into the inguinal canal, and corresponding minutely to the terminal fiber of the round ligament. In Hecker's[18] case there was removed from a sterile woman of forty years a tumor of the right labium majus, which upon examination proved to be a myoma of the right round ligament, associated with an ovarian hernia. Weber[19] cites a case in which there was found a fibrolymphangioma arising from the left round ligament, forcing its way down the greatly dilated inguinal canal, and finally becoming pedunculated, and hanging free between the legs. This occurred in a young virgin.

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[13] Quoted by Fromme.

[14] Quoted by Fromme.

[15] Boston Med. and Surg. Jour., 1864, lxxi, 478.

[16] Archiv. f. Gyn., 1883, xxi, 279.

Many other instances might be cited in which the round ligament was clearly the seat of these tumors. In my own case there was no evidence of such formation, and the tumor is classified as one of the group springing from the subcutaneous connective tissue.

Fromme is authority for the statement that fibromyxomas and pure myxomas of the labia should never be considered as primary growths developing from mucous membranes, but always as secondary degeneration forms of fibromas or myxomas. Examination of my specimen showed it to be one of this class.

THE TREATMENT OF DIFFUSE SUPPURATIVE PERITONITIS.[1]

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In this communication I shall discuss only the operative and postoperative treatment, not the preventive treatment, of diffuse peritonitis. In regard to the degree of peritonitis in the cases to be reported, I may say that only those with a definite purulent exudate extending throughout at least the greater part of the peritoneal cavity, and accompanied with definite signs of diffuse, undefined peritoneal inflammation, have been considered. Cases with a moderate amount of seropurulent exudate in the vicinity of the causative inflammation or injury are not included. The extent of the exudate has generally been estimated by the return flow through the irrigating tube used to wash out the cavity, which method, as will be explained later, has been found to be reliable.

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[17] Monatssch. f. Gyn., 1903, lvii, 441.

[18] Inaug. Diss. 1882 (quoted by Nebesky).

[19] Monatssch. f. Gyn., 1899, ix, 591.

[1] Read at a meeting of the Chicago Medical Society, November 21, 1906.

Through the courtesy of Drs. Hotchkiss, Peck, and Martin, who have been using, at the Roosevelt Hospital, essentially the same methods as myself, I am enabled to add their cases to mine, and can report a much larger and consequently more valuable series than if restricted to my own.

The series includes 78 cases of diffuse peritonitis due to appendicitis, contributed by Drs. L. W. Hotchkiss, C. H. Peck, W. Martin, and myself; 13 due to the perforation of gastric and duodenal ulcers and to traumatic perforations of the upper intestine, all with extravasation of gastric or intestinal contents, contributed by Dr. Peck and myself; and 8 cases of my own, caused by the perforation of typhoid ulcers. The series includes all the cases occurring in our services since the treatment to be described was begun, but does not include earlier cases treated by other methods. I have added my cases of typhoid perforation to the series because they prove that good results may be obtained by these methods, although, as a rule, they are unsuitable for the comparison of the methods of treatment of peritonitis.

The treatment of diffuse peritonitis may be divided into two phases: the operative and the postoperative treatment. The principles of our operative treatment are as follows: (1) To remove, as rapidly as possible, through a small incision, the origin of the inflammation, (2) to wash or irrigate the peritoneal cavity, and (3) to use as little drainage as possible, and not to attempt to drain the general peritoneal cavity. I think no one disputes the necessity of a rapid operation. The lessened time diminishes shock and the additional toxemia and irritation of the respiratory tract caused by the anesthetic.

The desirability of washing out the peritoneal cavity is disputed, the chief objection to my mind being the additional time required. In a few cases it may be carried out while the appendix is being removed or a perforation closed. In most instances, however, this is impracticable and the washing constitutes a distinct part of the operation. Even then, with the proper apparatus, it can be accomplished in from three to five minutes, and does not unduly prolong the operation. Another objection raised by many is that it entails additional traumatism. We have not found this to be true. The double-current irrigator works at its best through a small incision and does not injure the peritoneum.

The imperative indication for washing, to my mind, is the presence of extravasated gastric or intestinal contents in the peritoneum, for it seems to me at least undesirable to leave such foreign material. We might as well leave it in an ordinary wound. In our 13 cases of gastric perforation, food remnants in varying amounts were removed with the irrigator from remote portions of the abdomen. This matter, if left in the abdomen, would undoubtedly act as foci for the maintenance of the diffuse peritonitis or for the institution of local abscesses. Washing with the double irrigator removes this matter rapidly and, apparently, thoroughly, and with much less damage and shock than by any other method of cleansing. Our results in these cases fairly prove this point.

Of the 13 patients, the only ones that died of peritonitis were 3 in whom perforation had existed for over thirty-six hours, the only other death in the series being due to intestinal obstruction from adhesions on the twenty-sixth day after operation. Recovery also ensued in 4 out of 8 cases of typhoid perforation treated by me in this way. These results, I think, prove that irrigation is at least useful in cases of extravasation of gastrointestinal contents.

I have elsewhere described the irrigator[2] we have used in these cases, and at present it is only necessary to say that its efficiency is largely due to the fact that the fluid is removed by siphonage as rapidly as it is introduced, so that a peritoneal pocket or fossa may be rapidly washed out without diffusing the pus or other matter, and then the instrument is passed into another region and the whole peritoneum is thus systematically cleansed. All parts of the cavity can be reached through a small incision, thus avoiding large incisions and evisceration. Washing in this way accomplishes quickly what is more slowly and imperfectly performed by drainage of the general cavity. In other words, lavage may be considered as rapid drainage. Lavage completed, the usually small wound is rapidly closed with a few stitches of catgut, leaving a small opening only for drainage. The drain, unless deep drainage is indicated, simply passes through the wound and just enters the abdominal cavity, thus allowing the excess of the irrigating fluid to escape and preventing suppuration of the wound, which usually occurs if it is omitted. If any indication for deep drainage exists, such as imperfect removal of the cause of the peritonitis, unsafe closure of perforations, necrotic material or oozing, a suitable drain is carried to the site just as would be done if no diffuse peritonitis existed. The patient is then returned to bed in the Fowler position, and copious rectal salines are used. The salines are administered in different ways according to the indications. I am much in favor of an intermittent irrigation of the pelvic colon through two long rubber tubes, the inlet tube being smaller than the other. These tubes are left in place, and every three or four hours during the first forty-eight hours or so a slow irrigation, lasting from thirty to forty minutes, is carried out, the temperature of the fluid used being from 110° to 115° F. Large quantities are absorbed in this way, gas is removed, and the heat not only stimulates the patient generally, but the intestines also, and promotes peristalsis. We have also used the constant slow method advocated by Dr. Murphy with much success. The stomach is usually washed out on the table and no food is given until peristalsis is re-established. Morphine is given immediately after the operation to give rest, but not after the first twenty-four hours. Early catharsis is not employed. The Fowler position is used not for drainage purpose, but because it seems to enhance the patient's comfort and diminish vomiting.

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[2] Surg., Gyn., and Obst., May, 1906.

If we compare the treatment outlined with the very successful treatment recommended by Dr. Murphy, of Chicago, we find that it is exactly the same in all details excepting the manner in which the septic material is removed from the peritoneal cavity, and consequently the advantages of one method over the other, if any, depend upon the relative advantages of peritoneal lavage and general peritoneal drainage. These have been discussed many times, and some of the discussions are most interesting, even if some of the arguments and conclusions are fallacious.

In the first place, we must take it for granted that the improvement in the results of the treatment of this class of cases has been general, and has not been so dependent upon whether lavage or general drainage has been employed as upon the increased rapidity in getting the patient off the table, the use of large quantities of fluid by the bowel, rest for the entire gastrointestinal tract, and possibly the Fowler position. Still we should leave no stone unturned in our endeavor to save these desperate cases, and so I may be pardoned if I advance my views in favor of washing *versus* drainage, if for no other purpose than to provoke discussion.

I have already mentioned, as one of these advantages, the removal of foreign matter other than the peritoneal exudate. I also think that the septic products of bacterial growth can be more securely eliminated by lavage than by drainage. In regard to this point, it seems to me that the physiological and physical properties of the peritoneum are usually misunderstood. There seems to be a prevailing impression that soluble substances mainly escape from the peritoneum directly into the lymphatic vessels by a sort of filtration. This assumption, it seems to me, is rendered improbable if not disproved by the investigations of Orlow and Adler and Meltzer,[3] which show that absorption from the peritoneum is almost entirely by osmosis. For example, the absorption of isotonic salt solution is very much slower than of solutions of lower concentration. It is extremely probable that the converse is also true in peritoneal inflammation, namely, that the formation of toxic substances produces concentration of the peritoneal fluids and causes osmosis of the fluids from the tissues into the peritoneum, thus accounting for the accumulation of fluid exudate. In any event, the supposition that under certain conditions, as when the tonicity of the blood is decreased by saline infusions or by rectal absorption, a reverse current is established in the lymphatics and toxic substances are washed from the "absorbents" into the peritoneum, thus enabling them to be removed by drainage, is untenable. Under such conditions the blood and tissue fluids become hypotonic to the peritoneal exudate and a definite interchange is instituted according to the laws of osmosis, and the water in the blood and tissues passes into the peritoneal cavity, and substances in solution, that is, toxins, etc., pass, to a certain extent, into the tissues and vessels. Consequently, dilution of the blood, while the peritoneal exudate is left in a condition of septic concentration, must be a source of danger. In other words, when a peritoneum filled with septic exudate is simply drained, the increased flow of drainage noted, when large quantities of fluid are given by the rectum, represents, to a great degree, so much innocuous water, while the septic products are absorbed into the circulation to be eliminated by the kidneys and other excretory organs. On the other hand, if the septic products are removed from the peritoneum by irrigation, with a hypotonic saline solution, such as the ordinary 0.6 per cent. solution, and a certain amount is left in, an osmosis is at once instituted by which the water is absorbed from the peritoneum by the tissue and blood, while the septic products present remain in the peritoneal cavity, and consequently septic absorption, for the time being, is arrested. That there is an arrest of absorption is evidenced by the drop of temperature observed immediately after operation, except in cases that are already generally septic. The danger of disseminating the infection by lavage is, I think, overestimated. The process in these cases is already diffuse, and irrigation with a double-current instrument is not likely to spread it farther. Irrigation, of course, should not be done in local peritonitis, particularly when the limiting adhesions have been broken up, sponging being sufficient in these cases. Even if a few bacteria are carried by the washing solution to remote portions of the peritoneum, they are quickly taken care of by the phagocytes, which, although stimulated and increased, have not, as yet, suffered in vitality. As we well know, pus often flows over the healthy peritoneum when an abscess is opened without provoking inflammation. Further, it must be remembered that the exudate has no opsonic power and that the phagocytes in it are satiated or killed. Its rapid removal by washing allows an escape of active fluids and cells which will not be neutralized by toxic substances in the exudate. Even if saline solution is left in the abdomen, its absorption is so rapid that little is to be feared from its diluting protective substances.

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[3] Jour. Exper. Med., 1896, vol. i, No. 3.

In regard to drainage, if one, after irrigating, can feel assured that he has left nothing in the peritoneum but what can be absorbed, there is no need of drainage. If he has left something which probably will not be absorbed, he should drain, but he should only drain to the doubtful substance. Of course, one drains to an insecure spot; the drain is then a sort of safety valve.

We are all familiar with Dr. Ochsner's method of localizing and preventing diffuse peritonitis. Under it, undoubtedly, the outlying zones of moderate peritoneal inflammation that are filled with a seropurulent exudate undergo resolution and the exudate is absorbed. In many cases of appendicitis there is a fairly active exudation of seropurulent fluid in the vicinity, but without gross perforation of the appendix. With the removal of the appendix the fluid is rapidly absorbed. Such cases would often, in the ordinary course of events, go on to a definite diffuse peritonitis; yet, with the cause removed, they resolve, and they resolve without washing or drainage. When, in diffuse peritonitis, drainage of the entire cavity is attempted at the most dependent part, there must be portions which are not entirely emptied of the septic fluids, but these fluids become absorbed. There is ample proof, in other words, that the peritoneum will take care of considerable amounts of septic material, and we have found that after irrigation what little septic material is left is readily taken care of without the need of drainage. If, by accident or otherwise, material sufficient to cause continued suppuration has been left it has produced local abscesses, and these have been successfully opened later on. These secondary local abscesses have not occurred more frequently in cases not drained than in those drained. In the 99 cases there were 4 in which secondary abscesses occurred during convalescence, 2 in the undrained and 2 in the drained.

A patient not included in the series, and still in the hospital, was not irrigated but drained to the stump of the appendix and to the pelvis, through a stab wound over the pubes. In spite of the Fowler position, this patient developed an abscess between the liver and the hepatic flexure of the colon, on the fourteenth day after operation. From this it is evident that the manner of the treatment has not had much bearing upon the occurrence of secondary abscesses.

In comparing our fatal cases, I have also found that the employment of drainage, apparently, has had little influence on the results. Of the 78 cases of diffuse peritonitis caused by appendicitis death occurred in 15, a mortality of 19.2 per cent. Of the patients who died, 7 were not drained and 8 were drained, 7 to the stump of the appendix and 1 to the pelvis; 2 of the patients who were not drained recovered from the peritonitis, 1 dying on the sixth day, the cause not being determined, and 1 on the fifth day from double pneumonia, the autopsy showing that the peritonitis had resolved; 2 of the patients who were not drained and 2 who were drained were practically moribund and died within a few hours. The remaining deaths were due to sepsis and continuation of the peritonitis. The mortality percentage may seem large, but it must be remembered that these are unselected cases occurring in a very active ambulance service in one of the worst districts of the city, and that our policy has been not to refuse the hope derived from an operation. Of the patients with appendicitis who recovered, 31 were not drained, 28 were drained to the stump of the appendix, and 4 were drained to the pelvis. Of the 13 patients with peritonitis caused by perforations or ruptures of the stomach and upper intestine, 4 died, a mortality of 30.7 per cent. Of those who died, 2 were not drained and 2 were drained to the suture line in the stomach. One of the deaths in the not drained was due to intestinal obstruction from adhesions on the twenty-sixth day after operation, the peritonitis having resolved. In this case, pylorectomy had to be done to get rid of the ulcer, as it was impossible to suture it. The other 3 deaths occurred in patients who had perforated over thirty-six hours before operation. Of the 9 recoveries in the stomach series, 4 were not drained, 4 were drained to the suture line in the stomach, and 1 was drained to the pelvis. Of the 8 patients with perforation of typhoid ulcers, 4 died, of whom 3 were drained and 1 was drained to an abscess in the pelvis. Of the typhoid patients who recovered, 2 were not drained and 2 were drained to the pelvis, there being abscesses present. In the typhoid cases the head of the bed was only slightly raised.

In explanation, I may say that the abdominal wounds were drained in practically all, and when a case is stated as not to have been drained it refers to the peritoneal cavity. When drains have been passed to the site of the appendix or to the suture line in the stomach, in cases of perforation, of course, local drainage alone was instituted and not general drainage. In the few instances in which drains were passed to the pelvis, the peritoneum may be considered to have been generally drained, although the drains were not inserted for that pupose.

In conclusion, I may say that one cannot wholly estimate the success of a therapeutic procedure by statistical tables. Personal impressions seem to me to be more reliable if derived from sufficient observation. It is my belief that our patients have made smoother recoveries by the omission of drainage when possible, and I cannot look back with regret upon an occasion when I have omitted it. On the other hand, it is impossible to draw hard-and-fast lines as to the method of treatment of these cases, and judgment must be continually exercised. Some need drainage, some do not; some seem to do better with irrigation, others get well without irrigation or drainage; and some die whether drained or not, washed or unwashed.

REVIEWS.

The Practice of Obstetrics. In Original Contributions by American Authors. Edited by Reuben Peterson, A.B., M.D., Professor of Obstetrics and Gynecology in the University of Michigan. Pp. 1087, 523 engravings, and 30 full-page plates. Philadelphia and New York: Lea Brothers & Co., 1907.

In the preface we are told that this good-sized volume is designed above all to be practical, without neglect to theory. It opens with a section on the physiology and development of the ovum, by Dr. Huber, of the University of Michigan, who gives a clear and concise account of the subject from the standpoint of modern research. In stating the length of the embryo, the table commonly ascribed to Dührssen is credited to Hasse. In describing the decidua, the older terms, serotina and reflexa, are retained. The syncytium and Langhans' layer of cells are attributed to the fœtal ectoderm. The placenta is said to contain both a fœtal and maternal circulation.

The physiology of pregnancy receives treatment by Dr. Manton, of Detroit. The more modern view that the maternal heart is not hypertrophied during pregnancy is accepted as established. In treating of the urinary system during pregnancy, it is stated that the urea and solid constituents of the urine remain practically unchanged, but subject to normal variations. This has not been our experience, as we rarely see a pregnant patient in whose urine the solid constituents are not considerably altered in composition and quantity. No mention is made of altered function in the liver or other important organs connected with metabolism during pregnancy. Nausea and vomiting are considered as among the usual symptoms of pregnancy. In the next chapter, by the same author, on the diagnosis of pregnancy, vomiting is said to take place in only 49 per cent. of pregnant women. On the following page vomiting is recognized as sometimes due to toxemia, and as a symptom of importance. Perverted appetite is not, however, considered uncommon. The mammary changes caused by pregnancy are described in detail, and well illustrated. In diagnosticating pregnancy, external ballottement is regarded a symptom of value. This has not been our experience.

The physiology of labor is described by Dorland in a paper which is considerably illustrated. The old nomenclature of the various mechanisms of labor is retained, and the old divisions into four essential positions. Many of the phenomena of labor and its management are illustrated by photographs taken in the Maternity Department of the University of Michigan, with which the writer is not connected. It is usual for authors personally to superintend the preparation of photographic illustrations. The author takes occasion to warn, at some length, against the use of ergot during labor, a caution which we had supposed was scarcely necessary.

The physiology of the puerperal state is treated by Dr. Lewis, who advises that the cervix be exposed and lacerations sutured, should hemorrhage occur while the uterine body is firmly contracted. We have found it more advantageous to inspect the cervix and to close all considerable lacerations without waiting for hemorrhage. In considering the patient's posture and length of time in bed, it is said that danger of air embolism from turning is practically over in six hours after labor. It is questionable whether air embolism causes sudden death after labor, and the weight of evidence is in favor of the belief that such accident arises from cardiac clot, usually in the right side of the heart. The author wisely urges against the use of the catheter in the puerperal period if it can be avoided, and describes fully the antiseptic precautions necessary for such employment. We agree fully with him in the statement that ergot is the best means for preventing after-pains. Combined with strychnine it is of especial value. The author, however, urges against the routine use of ergot during the puerperal period. We believe that small doses of ergot, combined with strychnine, are beneficial to the majority of puerperal patients, producing good involution without injurious effect. Massage for puerperal patients is described at some length, and the knee-chest posture is advised. The necessity for thorough examination of all puerperal patients is urged.

Lactation is considered fully by Bacon, and the subject illustrated. In performing massage, he does not rub from the periphery toward the nipple, but commences in the axilla and under the clavicle. He employs alcohol freely upon the nipple to prevent infection.

The pathology of pregnancy is described by Ehrenfest, who discusses at length the toxemia of pregnancy. He does not ascribe much importance to the study of nitrogenous metabolism, but would confine his attention to the action of the kidneys. In treating the pernicious nausea of gestation, his choice of nutrient enemas has not been that most successful in our experience. Under disorders of the osseous system there is no mention of rachitis.

Considerable space is given to the pathology of the placenta, the decidua, the chorion, amnion, umbilical cord, and the fœtus, by Warthin, of Ann Arbor. These chapters are full, interesting, and well illustrated. Much-needed directions are introduced for performing an autopsy upon the fœtus or the newborn child.

Ehrenfest writes of the premature expulsion of the ovum, and urges the digital removal of the ovum when an operation is inevitable.

The dull curette is sometimes a useful adjunct. In criminal abortion, he properly warns against the employment of the sharp curette.

Schenck writes of extrauterine pregnancy, giving the diagnosis of the condition very fully. Immediately after rupture, operation by abdominal section is urged. In long-standing cases with hematocele, vaginal section is preferred. The pathology of labor is opened by a chapter on dystocia due to anomalies of expulsive forces. This gives place for a chapter by Ehrenfest, upon pelvimetry, which would seem to indicate that the measurement of the pelvis is a procedure to be employed in pathological conditions only. The reasons are not apparent why this chapter was omitted from the complete diagnosis and examination of pregnant women. Moran, in a well-illustrated article, writes upon labor complicated by anomalies of the bony pelvis. In discussing treatment, he recognizes the fact that Cesarean section is successful in proportion to the uninfected condition of the patient. Section should not be done on patients repeatedly examined when the most rigid antiseptic precautions have not been taken. He states confidently that when the conjugata vera is above 9 cm. symphysiotomy and Cesarean section should not be considered. This is not invariably the case, as the elective Cesarean section may be necessary under these conditions. Dystocia due to anomalies in the fœtus and its presentation or development also receives attention by Moran.

Moran writes at length upon eclampsia, and takes the ground very properly we think, that care should be taken to avoid violence in efforts at delivery.

The pathology of the puerperal state is described by Lewis, who advises starvation and the use of cold externally for appendicitis. When the uterus is displaced during convalescence, if subinvolution is present, curetting should be done. The knee-chest posture is always indicated; tampons and pessaries may also be employed.

Puerperal infection, also, is described by Lewis, who recognizes the danger of bath-water as a source of infection. The uterine pathology in infection is minutely described, and methods of examination for puerperal infection are also given. Uterine douches are sometimes indicated, and the blunt curette may also be used. Salt solution is valuable, but antistreptococcic serum has proved unsatisfactory.

Crockett writes upon the obstetric operations. Care is taken to illustrate well-fitting and ill-fitting rubber gloves, and stress is laid upon their employment. Curetting and gauze packing are described, and the use of salt solution by transfusion is fully illustrated. The induction of premature labor is described by the same author, with photographs and reproductions which are familiar. In treating lacerations, no mention is made of laceration of the anterior segment of the pelvic floor and the tissues about the urethra. This accident occurs not infrequently and always demands attention. In the chapter on forceps, by the same author, no axis-traction forceps but Tarnier's is mentioned. We agree with the writer in cautioning against the use of forceps when the head is not engaged in the pelvis.

In treating of Cesarean section, his nomenclature is peculiar. He describes the abdominal conservative operation, the abdominal Porro operation, panhysterectomy, and vaginal section. He confuses abdominal celiohysterectomy with intraperitoneal treatment of the stump with the original Porro operation.

Lewis writes of the care of the newborn; Bacon of premature infants; Lewis of artificial feeding, concluding the volume with a chapter on the diseases of the newborn. In describing by illustration the so-called Byrd-Dewey method of resuscitating a newborn infant, the child is not held with the head downward; this is a matter of some importance, as mucus is thus more readily expelled from the child's mouth. Schultze's swinging method is illustrated, but unfortunately with imperfect photographs.

The volume contains much of interest, is clearly printed, and has among its illustrations some excellent photographs and some clear and good cuts. The illustrations, however, are not of uniform excellence. E. P. D.

Abdominal Operations. By B. G. A. Moynihan, M.S. (Lond.), F.R.C.S., of Leeds, England. Second edition. Pp. 815. Philadelphia and London: W. B. Saunders & Co., 1906.

The best indication of the value of this work is the fact that a second edition has been called for within a year. The changes, of course, are not very extensive, but a number of new illustrations are to be found, as well as certain changes in the text. The extent of the work, as relates to subjects, however, has not been increased. The author does not include any of the gynecological operations, nor those on the kidney, ureter, or bladder. He also has excluded hernia. We think that the omission of the various operations for the different forms of abdominal hernia is to be regretted. The author does not undertake to describe all the operations for the different pathological conditions found in the abdominal organs, but chooses those most in use and those which he has found practical. In his chapter on the treatment of general peritonitis we think that the omission of the Murphy method of operating in these cases is a mistake. Great attention is paid to the preparation of the patient for operation, and also to the after-treatment. Personally, we are not inclined to agree with the early feeding by the stomach which the author recommends after operations upon perforated gastric ulcers, believing that the longer food is kept out of the stomach the greater the chance for thorough healing of the ulcer, and the lessened likelihood of future operation. In dealing with the treatment of gastroptosis one is rather struck with the fact that the author does not recommend gastroenterostomy, which would appeal to us as a simpler method in treating this condition than that of trying to elevate the stomach to its original position with all the chances of future elongation of the adhesions and recurrence of the trouble.

The book is written in a comprehensive manner and is full of valuable practical advice to surgeons. The technique is always simple, as few assistants and as few mechanical aids as possible being advised. The author has a forceful and confident manner of expressing his views, and the simplicity of his style is one of the attractive features of the book. The illustrations are excellent, and the work is such a thorough and reliable guide to the operating surgeon that we believe it will rank as one of the best books of its kind.

J. H. G.

The Surgical Assistant. A Manual for Students, Practitioners, Hospital Internes, and Nurses. By Walter M. Brickner, B.S., M.D., of New York City. New York: International Journal of Surgery Company, 1906.

The Operating Room and the Patient. By Russell S. Fowler, M.D., Surgeon to the German Hospital, Brooklyn, New York. Pp. 172. Philadelphia and London: W. B. Saunders Company, 1906.

A Nurse's Guide for the Operating Room. By Nicholas Senn, M.D., Ph.D., LL.D., C.M., Professor of Surgery, Rush Medical College, in affiliation with the University of Chicago. Pp. 204. Second edition. Chicago: W. T. Keener & Co., 1906.

Surgical Suggestions: Practical Brevities in Diagnosis and Treatment. By Walter M. Brickner, M.D., Chief of the Surgical Department, Mount Sinai Hospital Dispensary; and Eli Moschcowitz, M.D., Assistant Physician, Mount Sinai Hospital Dispensary. Pp. 58. New York: Surgery Publishing Company, 1906.

The technique of present-day surgery constitutes no small or unimportant part of the art, and it is important that the assistants and nurses should understand its importance and practice it with as scrupulous care as the surgeon himself. This being true, there is a distinct place for such books as the above, and it is well they should be written by experienced operators.

Brickner's *Surgical Assistant* is somewhat new in its scope, but it fills a want which many surgeons have felt. It undertakes to show the assistant, whether he or she is a hospital interne, a general practitioner, or a nurse, what the duties of an assistant are and how to perform them. There can be no doubt, after the careful perusal of this small volume, that it is a book which may be studied with advantage by all hospital internes and nurses. The first chapter deals with the general conduct of the assistant in his relations to the surgeon and to the patient, and contains a great deal of good advice. The remaining chapters of Part I deal with the assistant's conduct during the preparation of a patient, the operation, and the immediate postoperative care of the patient. In Part II the various operations are taken up, and special attention is paid to the part the assistant plays in them. As an appendix the various preparations of surgical materials are considered and the various surgical instruments illustrated.

Fowler's *Operating Room and the Patient* may be accepted as a thorough and complete description of a modern operating room and a reliable guide to the preparation of the patient, instruments, dressings, sutures, etc., for the different operations. The last chapter is composed of lists of instruments required for operations on different parts of the body, and will be found useful to a clinical nurse or assistant preparing for an operation, especially if it is to be done outside of a hospital. The arrangement of the operating room, the preparation and the preservation of different materials needed, are given in detail. Under the subject of anesthesia we regret the omission of ethyl chloride as a general anesthetic. The use of ethyl bromide is described, but we do not think this agent is as deserving of confidence as the chloride, nor is it being so extensively employed. In describing the preparation of the patient for operations on the stomach no mention is made of the importance of cleansing the mouth for some days before operation. The different positions which the patient should occupy on the operating table are well illustrated, but there seems to be no necessity for picturing the patient in the "altogether."

Senn's *Nurse's Guide* is a second edition. Its basis is a course of lectures delivered to nurses. It is not as extensive as the two preceding works, but as it is written for nurses alone this can hardly be a fault. The various instruments used in surgery are illustrated, which must prove of value to the nurse.

Surgical Suggestions, by Brickner and Moschcowitz, is composed of terse statements regarding diagnosis and treatment of different diseases arranged anatomically. The following extracts illustrate very well the character of the text: "In seeking a cause for torticollis do not fail to examine the teeth." "Ascites in the presence of a mass in the pelvis usually, but not necessarily, means malignancy." This small volume is filled with other such rules which will be found of invaluable benefit to the general practitioner and the occasional operator. J. H. G.

DAS PROBLEM DER SYPHILIS. By PROF. DR. O. ROSENBACH. Berlin: August Hirschwald, 1906.

THIS book of 164 pages is a second edition, testifying to its cordial reception by the profession, for the thinking portion of whom it was written. Everywhere the subject is discussed in a broad and scientific manner worthy of so distinguished an author and practitioner. The subject matter is considered on distinctly original lines, as evidenced by the first chapter, which goes thoroughly into the question of "specific remedies." The conclusion is reached that experience teaches that the influence of the individuality upon the origin, development, natural course of the disease, as well as upon the forces influencing the disease, are all of the greatest significance. The syphilitic subject, laboring under the action of the specific virus, is to be regarded as a bearer of contagion only so long as ulcerative primary processes, diffuse eruptions, and moist papules are produced. The glandular swelling, which shows the existence of constitutional disease, is to be regarded merely as a sign of strong general reaction, and, in the later stages, as a remnant which generally terminates in spontaneous recovery. After the period of recent manifestations has passed it is neither an expression of the specific nature of the disease nor the seat of a specific virus. The author carries out these lines of thought still farther. Notwithstanding that in the majority of cases these stages of the disease may be distinguished, this chronological division should be set aside, inasmuch as, according to the individual proportions, the symptoms, which are the expression of a general disturbance of nutrition, may be precocious. For the sequels of syphilis the author takes the ground that specific treatment should be employed with reserve, inasmuch as mercury, so useful in the early eruptions, possesses no action as a prophylactic nor in preventing relapses. The treatment of local or constitutional diseases, and their sequels, in the author's sense, should follow the same principles as in the handling of all constitutional diseases characterized by secondary symptoms. The patient should be under the observation of the physician treating him for at least one year from the date of the first cutaneous symptoms; and here Dr. Rosenbach directs attention to the interpretation of the many cutaneous and other symptoms that during this period are liable to appear, and their proper treatment, as bearing upon not only the use of drugs but also of other methods of coping with the disease. The author sees much in syphilis beyond the bare symptoms which every general practitioner readily can see, and points the way to the proper care of the patient as being of much more importance than stereotyped medication. The book is replete with interesting and valuable reading matter, and in scope and style is quite unlike any other similar treatise on the subject. We should like to see the little volume translated into English.

L. A. D.

ATMOKAUSIS UND ZESTOKAUSIS: DIE BEHANDLUNG MIT HOCHGESPANTEN WASSERDAMPF IN DER GYNAEKOLOGIE. (ATMOKAUSIS AND ZESTOKAUSIS: THE GYNECOLOGICAL TREATMENT WITH STEAM AND HIGH PRESSURE). By DR. LUDWIG PINCUS. Second edition. Pp. 371. Wiesbaden: J. F. Bergmann, 1906.

THIS interesting monograph is an exhaustive review of the author's extended experience with a method of treatment which has received comparatively little attention in America, principally because of timidity and unfamiliarity with the technique, simple as the latter is. The first section of the first part of the book (pages 12 to 189 inclusive) is devoted to the history and development of the operation, including a reply to the various criticisms that have been urged against it. The following cautions are emphasized: (1) Thirty seconds should be regarded as the *maximum* time during which steam should be allowed to enter the uterine cavity. (2) The treatment must not be repeated until *complete regeneration of the endometrium has taken place.*

The second and third sections deal with the apparatus, and the proper method of using it, in order to avoid the sloughing and subsequent cicatrization of the soft parts of which many operators have complained.

An interesting chapter on experimental work is followed by one on anatomy, the latter being fully illustrated by photographs of uteri, showing the results obtained by the application of steam for varying periods of time, and the fact that its effects can be controlled by careful attention to the rules laid down.

The second half of the monograph is devoted to clinical facts, stress being laid upon the following points as necessary to the success of the treatment: thorough asepsis, thorough dilatation of the cervical canal and preliminary cleansing (*not curettement*) and drying of the uterine cavity, and the recognition of contraindications, especially disease of the adnexa. Narcosis is not necessary, as a rule. The treatment should not be attempted during menstruation, unless it is absolutely necessary to check serious hemorrhage. Strict rules are given with regard to keeping patients in bed under careful observation for not less than four days. The various indications and contraindications for atmokausis and zestokausis are given at length (pp. 229–239), so that the reader may infer that this method of treatment has been as carefully developed as the technique of any modern operation—if not more so. These have already frequently appeared in current literature and need not be repeated.

Preclimacteric bleeding is regarded as the most important indication for atmokausis, malignant disease being carefully excluded. The author states as a positive rule that total extirpation should never be performed for hemorrhage alone until atmokausis has had repeated trials. A number of successful cases is cited. The results in cases of hemophilia are stated to be especially gratifying. The precautions to be observed in cases of hemorrhage due to fibroids are worth careful attention. Submucous growths, irregular depressions in the uterine cavity, are to be regarded as positive contraindications. In fact, the writer is careful to give the impression that the treatment should be limited to cases of hemorrhage in which on account of great weakness or cardiac complications a radical operation is unjustifiable.

Menorrhagia and metrorrhagia in childbearing women, in whom hemostasis alone is indicated, is contrasted with cases in which actual obliteration of the uterine cavity is desired, as in women with incurable diseases. Special sections follow on the treatment of subinvolution, senile endometritis, membranous dysmenorrhœa, and chronic gonorrhœal endometritis, in each of which different precautions are to be observed. The concluding section is devoted to the limitations of zestokausis, the gist of which is that the zestocautery is to be regarded as a sort of mild thermocautery in which an eschar seldom results.

This monograph concludes with an exhaustive bibliography, no less than four hundred writers being quoted. We are sure that the American reader who recognizes the great care with which the author has developed his method of treatment, will be induced to lay aside his prejudices against it and accord it a fair trial.

H. C. C.

THE DISEASES OF THE NOSE, THROAT, AND EAR. By CHARLES PREVOST GRAYSON, A.M., M.D., Clinical Professor of Laryngology in the University of Pennsylvania. Pp. 550. Second edition. Philadelphia and New York: Lea Brothers & Co., 1906.

THIS book is not only a thorough revision of the previous edition, but shows very extensive additions and alterations to the text. It is intended primarily for the use of students and beginners in the practice of rhinology and laryngology, and in fulfilment of this aim its author has shown the result of his long experience as a teacher in the University of Pennsylvania. In clear, concise diction he gets at the gist of his subject and presents it in easily assimilable form. One especially valuable feature of the work is the emphasis laid upon the relation of local lesions in the nose and throat to constitutional disorders. In Chapter IV there will be found a particularly good discussion of this subject. The author may possibly be regarded as holding rather strenuous views on the importance of faulty metabolism in various pathological conditions in the upper respiratory tract, but he advances good sound logic in support of his tenets. His discussion of chronic rhinitis contains a very interesting, if somewhat lengthy, quotation from an article previously published by him on the relation borne by sexual excitements to nasal disorders. This subject is but seldom considered, and yet is undoubtedly one of great importance, as any rhinologist of experience can testify. In dealing with that mysterious symptom-complex, hay fever, the author very candidly states how unsatisfactory most of the loudly proclaimed methods of treatment have proved. He mentions, only to condemn, the treatment with so-called "liquor ambrosiæ," and he rather slyly intimates that "pollentine" may soon be cast into the limbo with the ambrosial fluid. Another strikingly good feature of the book is the section on laryngeal tuberculosis, a subject possessing great difficulty and too often handled merely in a routine manner. In conclusion, we would say that the book will certainly hold the position which it has already achieved as a first-class practical manual for the student or practitioner beginning the study of laryngology. F. R. P.

The Technique of Operations upon the Intestines and Stomach. By Alfred H. Gould, M.D., of Boston. Pp. 302. Philadelphia and London: W. B. Saunders Co., 1906.

This book contains the result of an experimental study of gastrointestinal technique, the object being to simplify as far as possible the best gastrointestinal operations. In addition to the original work which has been done the author has included much of that done by Harrington, Nichols, F. T. Murphy, Barbat, and Monks. The first portion of the book deals with the reparative process as it occurs in intestinal wounds after various methods of suture and mechanical appliances for performing anastomoses. It is very interesting and instructive, and is beautifully illustrated. The next portion has to do with suture materials and the method of their use. Here the various intestinal needles, sutures, and knots are described and illustrated. The third portion deals with the anatomy of the intestines, in which Monks is freely quoted and a number of illustrations from his article on intestinal localization employed. The latter portions of the book relate to operations upon the intestine and stomach. Here the different operations are carefully described and excellently illustrated. The illustrations throughout the book deserve especial commendation. We have never seen better. As a guide to technique and to the various steps of gastric and intestinal operations the book will be found most trustworthy. J. H. G.

PROGRESS

OF

MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF

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The Superficial Extension of the Percussion Shock.—HAMBURGER (*Münch. med. Woch.*, 1906, liii, 2283) lays great emphasis upon the importance of the chest wall in the production of percussion sounds. Between the early view of Williams, that the note arises entirely from vibration of the chest wall, and the later contention of Skoda, that the vibration of the air in the lungs is the sole factor, he accepts with Mazonns an intermediate position, looking to a combination of both as the only satisfactory manner of explaining percussion phenomena. The distinct dulling of resonance which occurs when an assistant, with his hands, compresses the chest wall on either side of the point percussed, appears to Hamburger definite evidence of the part played by the chest wall. The intensity of the percussion note depends upon the wave mass, as well as upon the wave amplitude, and the wave mass is determined by the amount of air in the lung being set in vibration. The percussion stroke is carried not only a certain depth into the lung, but radiates in a circle for a certain distance over the chest, the chest wall acting as a pleximeter. Weil was the first to demonstrate that relative liver dulness and heart dulness do not depend upon the dampening or dulling by the solid organ of the resonant note arising in the superimposed layer of air-containing tissue, but that it is due to the decreased vibrating mass over the solid organs. From experiments on corpses he demonstrated that dulness began at a point where the lung substance was 4 cm. in thickness. Weil took into consideration only the radiation of the percussion shock into the depth of the organ. Hamburger insists upon the equal importance of its superficial transmission. If our percussion note radiating from the point of origin is able to carry in all directions over the chest wall, say 6 cm., when we approach within 4 cm. of the border of the lung a considerable portion of the circle is cut off and the mass of vibrations equally diminished. The same explanation holds good for the frequency with which a tympanitic note is heard high up in the left axilla: in this instance the air in the stomach is thrown into vibrations. Upon the same basis is explained the areas of paravertebral dulness and paravertebral resonance in pleural effusions. The more elastic the chest wall the wider the area of superficial transmission. The practical importance of the principle is found especially in its application to children. Any position the child may take or any manner of holding it which will compress one side or a portion of a side may give rise to percussion differences which have no pathological basis.

The Pathogenesis of Acute Pancreatic Hemorrhage and Pancreatic Necrosis.—Palya (*Berl. klin. Woch.*, 1906, xliii, 1562), from a consideration of the views of Chiari and others of the self-digestive action in disease of the pancreas, had his attention drawn to an important factor which had been previously disregarded. The work of Pawlow and his pupils has proved that the pancreatic secretion itself is inactive and that it becomes efficient only when mixed with intestinal juice. This activating power resides in the substance Pawlow calls enterokinase, which in very small quantities can convert the trypsinogen into active trypsin. To determine the importance of the presence of this substance in the gland itself Palya performed four sets of experiments:

1. The injection of intestinal mucosa extracts. (*a*) In 2 animals the extract was injected directly into the substance of the gland. One was positive, 1 negative. (*b*) In 5 the centrifuged extract was injected intraparenchymatously. Three were positive; 2 negative. (*c*) In 3 the centrifuged extract was injected into the duct; 2 were positive, 2 negative. In 2 the centrifuged and filtered extract was injected into the duct; 2 were positive; 1 negative. (*d*) In 4 enterokinese prepared according to the directions of Bayliss and Starling was injected into the duct. All were negative. In the positive cases the animals died within twenty-four hours (1 in twenty-nine hours). Necrosis and hemorrhage of the pancreas and more or less widespread fat necrosis were found. In 3 cases there was simple swelling of the gland, but microscopically this also showed necrosis. Fat necrosis was marked in all but 1 animal, which died in five hours. Of the animals which recovered some were extremely ill and a few even moribund. Later laparotomies revealed a hardening and contraction of the gland, peripancreatic adhesions and, twice, small abscesses in the neighborhood of the duct. That these effects were due to the extract and not to bacteria which may have been introduced is indicated by the facts that fat necrosis seldom follows injection, that the period between injection and death was so short, that the anatomical changes were not those of a bacterial infection, and that the fat necrosis occurred even with the carefully filtered extract. The action of bacterial toxins or of bacterial kinese cannot be definitely ruled out. The entire absence of reaction after injections of enterokinase would seem to reject the supposed activating influence of the extract, but it is possible that the particular

preparation may have been faultily prepared or the presence of too large a quantity of enterokinase may have frustrated its own activity.

2. The injection of intestinal contents. Seven dogs received from 1 to 3.5 c.c. of duodenal contents into the pancreatic duct. In from twenty to thirty hours all succumbed, and all showed most extensive fat necrosis. In 4 the pancreas was the seat of extensive hemorrhage. In the other cases there was simple swelling of the gland, but microscopically all steps from simple parenchymatous degeneration to complete necrosis were observed.

3. In one instance 2.5 c.c. of the filtrate of a colon culture was injected into the duct. The animal after a few days' illness recovered, while a control which received the same subcutaneously died after a few hours.

4. Trypsin experiments. Seven animals were injected with solutions of trypsin and all proved positive. The lesions were as in the other experiments.

The most satisfactory results and the most extensive lesions were obtained with duodenal contents. As explained under the experiments with intestinal mucosa extract, it is hardly possible that the bacteria present could be responsible for the lesion. Nor can the oil, blood, bile, fatty acids or other substances, all of which are present in very small quantities, be an important factor in the light of previous experiments by numerous authors. It is known that a very small amount of enterokinase will activate a large amount of pancreatic secretion, and the uniform positive results following the injections of trypsin indicate that the mode of action of the duodenal contents is by causing such a conversion.

An Experimental Investigation of Sahli's Desmoid Reaction.—Saito (*Berl. klin. Woch.*, 1906, xliii, 1305). Sahli has suggested a method of ascertaining the functional efficiency of the stomach, depending upon its ability to digest catgut. Small rubber bags are filled with methylene blue or salicylic acid or iodoform and tied with catgut strings. One of these is administered after a meal and the time ascertained when the substance appears in the urine or sputum. Alexander and Schlesinger have shown that various substances are excreted by the same patient in different lengths of time, and in making comparisons a given substance must be constantly used. In disturbed gastric activity there is a distinct lengthening of the reaction time, most noticeable in subacidity or anacidity. Three factors are of importance in the reaction: (1) The solution of the catgut; (2) absorption of the drug; (3) its excretion. The third factor Saito allows to be constant in normal individuals, and proceeds to an examination of the other two. By a large number of experiments he shows that although catgut is not digested by pure pancreas secretion nor by pancreas secretion and gastric juice, nor by gastric juice and intestinal secretion, yet the mixture of pancreas secretion and intestinal extract will dissolve it in from six to twenty-seven hours. In cases of lost gastric secretion the desmoid, therefore, may still appear. Allowing that in a given case the catgut is dissolved in the stomach, there are other factors which may interfere with the correct interpretation of results. Saito shows that the rapidity of solution of the catgut depends upon the concentration of the digesting fluid, and this varies with the amount of food taken, the quantity of juice secreted, and the mechanical mixing power of the stomach. These factors, of course, are very variable and are difficult to control. In regard to the absorption of the drug he shows that methylene blue and iodoform are taken up only in the intestine. They must be discharged from the stomach before the reaction can appear. The motor function of the stomach, therefore, is an important factor. He concludes that the desmoid test is influenced by so many uncontrollable factors that it is of little use in diagnosis.

The Improvement Caused in Leukemia by Intercurrent Infections.—Funck (*Berl. klin. Woch.*, 1906, xliii, 1308) reports a case of myeloid leukemia with attacks of renal colic caused by the impaction of masses of uric acid crystals in the ureter. The blood was examined with care and particular emphasis laid upon the differential enumeration of the polymorphonuclear cells according to the method of Arneth. Arneth classes the polymorphonuclear cells according to their age; the more lobules the nucleus possesses the older the cell. Those with only indented nuclei he puts in Group I; those with two lobules, in Group II, and so on. He thinks that the ripe leukocytes are the ones that succumb first to an infection and by their disintegration supply antibodies. The patient, a young man, aged twenty-eight years, showed at his first visit an enlarged spleen, slight swelling of the cervical, submaxillary, and inguinal glands, and with the fluoroscope enlarged bronchial glands. The hemoglobin was 48 per cent.; the red cells, 2,930,000 with 5 per cent. of erythroblasts; the leukocytes 86,000, of which 48 per cent. were neutrophile; 28 per cent. eosinophiles; 11 per cent. mastzellen; 13 per cent. lymphocytes. 22 per cent. of the neutrophiles were myelocytes. Under *x*-ray treatment the leukocytes fell after one and a half months to 12,800, and the neutrophiles showed a marked improvement according to Arneth's definition. There were then 84 per cent. of neutrophiles, of which only 3 per cent. were myelocytes. The polymorphonuclear leukocytes of the first class fell from 57 to 11 per cent.; of the second rose from 37 to 52 per cent.; of the third, from 5 to 33 per cent.; of the fourth, from 0 to 4 per cent. *X*-ray treatment was then stopped and under arsenic improvement continued. On May 26, two months after he was seen, the patient had an attack of colic associated with infection of the renal pelvis. During the four days after the attack the number of leukocytes rose, and varied from 22,000 to 26,800. On the fifth day they fell to 10,600; on the sixth to 4200; on the seventh to 5000. On the tenth day they again went to 10,800 and continued over 10,000. In a table Funck furnishes, great improvement is shown during the rise in the number of white blood corpuscles, the neutrophiles rising, the myelocytes falling, and the polymorphonuclear cells going into the higher classes. With the drop in the number of leukocytes, however, is shown a most remarkable fall in the number of neutrophile cells, a drop from 86 per cent. to 12 per cent., and an equally remarkable rise in percentage of myelocytes from 0 to 82 per cent. At the height of the leukocytic reaction all polymorphonuclear cells but those of Class I had disappeared from the blood, Class IV dropping from 61 per cent. on the fourth day to 0 per cent. on the fifth day. When the infection subsided the blood returned to its previous condition, the neutrophiles rising to 88 per cent., the myelocytes falling to 4 per cent., the polymorphonuclears counting Class I, 12; Class II, 65; Class III, 22; Class V, 1. During the infection the erythroblasts rose to 22 per cent. of the red cells, to again disappear from the blood as it subsided. As is usual in such instances there was a marked decrease in size of the spleen and glands during the reaction. This picture corresponds closely to those usually observed, the infection being accompanied by marked improvement in the number and class of leukocytes, the improvement lasting a variable time and then returning slowly to the condition present before infection. The interesting portion of Funck's table is the remarkable swing to the left in the quality of the cells, while the number of leukocytes is lowest. His explanation of these conditions is this: He considers the bone-marrow too seriously diseased to furnish the great number of polymorphonuclear neutrophiles that flood the blood as the first reaction to the infection. They must come, he believes, from the myeloid areas in the spleen and glands, the sudden discharge accounting for the diminution in size of these organs. This supply, however, is very limited and as the older cells succumb to the infection and the bone-marrow is unable to respond with ripe forms, there is a great diminution in the number of cells, young forms remaining and being added to from the marrow. Funck warns that the number of leukocytes alone is no indication of the condition of the blood. Only a careful morphological analysis can give an idea of its quality. On the days when the count was lowest the patient's blood was in worse condition than when the count was higher.

Tuberculin Treatment of Pulmonary Tuberculosis.—HAMMER (*Wien. med. Woch.*, 1903, liii, 2343) for six years has employed tuberculin in the treatment of ambulatory cases of tuberculosis, and he is confident that its use has been followed by benefit in every instance. There is a tendency at present to restrict its use to treatment of early cases, and while most striking and lasting results have been obtained in this class of cases, Hammer believes that when judiciously used it may be applied with good results in practically all cases. His own material has been, on the whole, unfavorable, drawn from the poorer classes, and embracing advanced, as well as early and moderately severe, cases. He admits, that in a disease which has such protean manifestations as pulmonary tuberculosis, one must judge cautiously of the influence of a given remedy. The patients had not the advantage of good hygiene and diet. He points out that the one important element in success is to individualize; to carry the patient along carefully on a dose just short of the one able to produce a reaction. He has seen a marked general reaction occur after the administration of the 0.002 milligram. The curative action or increased sensitiveness to the drug must be carefully watched. As a rule, he begins with 0.01 milligram; in many cases, however, with 0.001 milligram. This dose is gradually increased until a gram of pure tuberculin is given. Such a course of treatment lasts, as a rule, six months, the injections being given at first twice a week, later once a week. The dose is advanced very slowly and cautiously, the patient being carefully watched, and at the occurrence of any suggestive subjective symptoms or slight temperature elevation the same dose is repeated or a still smaller dose returned to and a careful ascent again begun. Among the early closed cases Hammer has obtained satisfactory cures; many of the more advanced cases have become stationary, and even in the far advanced there has been a distinct alleviation of symptoms.

The Pathology of Polycythemia Rubra.—Glaessner (*Wien. klin. Woch.*, 1906, xix, 1475) reports the occurrence of polycythemia in a man aged forty-four years with tabes. The symptoms for which he sought relief were fulness in the head, headache, shortness of breath, distention of the abdomen, and swelling of the legs. For two years he had himself noticed an increasing redness of the face. He was addicted to alcohol and had had syphilis. There was most intense cyanosis of the skin and mucous membranes, and over the shoulders and buttocks numerous small purpuric spots. The skin over the chest and abdomen was covered with dilated venules. The liver and spleen were both markedly enlarged. Argyll-Robertson pupil and Romberg's sign were present. The blood count on admission was 10,000,000 red cells, 7000 leukocytes, and hemoglobin, 90 per cent. Morphologically, the red cells were normal, except for slight microcytosis and macrocytosis; the leukocytes were present in the usual proportion. Death occurred slowly from cardiac dilatation. The most striking feature at autopsy was the enormous quantity of blood in all parts of the body, not distributed to groups of organs or to a single region, as is common in chronic passive congestion. In the long bones, besides the yellow marrow, there were small areas bluish red in color, showing microscopically a great increase in the number of normoblasts, the leukocytes remaining normal in appearance and in relative proportion. The spleen was much enlarged, firm, and of a deep-red color. Scattered through its substance were numerous small, yellowish-gray nodules, the average about 1 to 2 mm. in diameter. At first appearance these resembled tubercles, but microscopically they consisted of areas of anemia and connective tissue overgrowth. In several of the instances of reported polycythemia a primary tuberculosis of the spleen was found at autopsy. Glaessner considers such an infection secondary and warns against making an error in diagnosis by mistaking the areas above referred to. He thinks the essential and distinctive lesion is the marrow change, the great increase in nucleated red cells. This will aid to distinguish many cases of pseudopolycythemia, which clinically may present the three cardinal symptoms: cyanosis, increased number of red cells, and enlarged spleen. Glaessner quotes a case of stasis in which the cause of obstruction during life was not apparent. The patient was very blue, and had a greatly enlarged spleen; the red cells were 9,600,000, and the hemoglobin, 120 per cent. At autopsy an adherent pericardium and chronic pleurisy were discovered as causes of the obstructed circulation.

SURGERY.

UNDER THE CHARGE OF
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A Simplification of Total Resection of the Bony Chest Wall in Empyema.—Bayer (*Zentralbl. f. Chir.*, 1906, xxxiii. 1) says that for the cure of neglected total empyema Schede's operation is certainly the most radical and surest method. Yet it must be admitted to be very severe for a patient much run down by long-continued suppuration. The hemorrhage resulting from the raising of a large flap of soft tissue is not unimportant. It not infrequently demands, from the occurrence of collapse, an abrupt termination of the operation and its completion at a second sitting. When on account of the unyielding character of the intercostal soft parts over the empyemic cavity it becomes necessary to remove them, the resulting mutilation is enormous. Bayer believes that the purpose of the Schede operation can be attained in very difficult cases by a more simple and conservative operation. A vertical incision about two fingers' breadth beyond the anterior axillary line will cross all the ribs from the third to the ninth or tenth, close to the anterior serrations of the serratus magnus muscle. It will not wound any of the large vessels and will divide no muscles except a few fibers of the external oblique from the seventh rib downward. About 2 cm. of each rib, beginning below at the ninth or tenth and going upward to the third, may be quickly removed subperiosteally with forceps. The intercostal soft tissues and pleura are then divided in this line with a thermocautery to prevent bleeding from the intercostal arteries. If necessary, the second rib may be removed by elevating the arm to displace the pectoralis major muscle, and extending the skin incision slightly. An incision over the lowest rib to be removed, extending to both sides of the original incision, will aid in the further steps of the operation and will provide for more posterior drainage at the conclusion of the operation. By separating the periosteum from the bone with a sharp raspatory, each rib can be stripped of its soft tissues easily, these then strongly retracted by an assistant, and the rib divided as far posteriorly as the angle and anteriorly as the junction of the bone and cartilage. The greater part of the chest wall is then free of the ribs. If it is then soft and flexible it sinks in spontaneously and the empyemic cavity disappears. If it is rigid, the costal pleura may be removed and if necessary transverse incisions in the chest wall made.

Bayer operated successfully on 2 chronic cases by this method after the simpler operations had repeatedly failed. In both he sutured only the upper angle of the wound and drained with a tube at the posterior end of the secondary incision. An abundant layer of gauze was laid over the approximated edges of the flaps of soft tissues and held in place by a bandage. In the second case, because of the rigidity of the ribless wall, it was necessary to make a curved incision in it before it would apply itself to the diaphragm and retracted lung. The especial advantages of this operation are that it can be done quickly without serious loss of blood, and that the resulting disfigurement is not great.

Drainage of the Prevesical Space through the Perineum in Suprapubic Cystotomy.—Belfield (*Annals of Surgery,* 1906, xlv, 101) says that while suprapubic cystotomy admittedly affords the best access for intravesical operations, the difficulty in drainage of the urine against gravity, when primary union is not obtained or attempted, leads to leakage into and infection of the prevesical space, commonly, in spite of the many devices to prevent it. The mortality of the suprapubic operation in elderly people is distinctly greater than that of perineal incisions for the same purpose, notably prostatectomy. He suggests draining the prevesical space into the perineum in the following manner: When the operator is ready to close the wound, the membranous urethra is opened on a grooved staff, the gorget introduced, and the staff withdrawn. A small trocar and cannula is passed from above along the anterior surface of the bladder and prostate into the groove of the gorget. The trocar being removed, a few strands of silkworm-gut are passed through the cannula along the gorget, out through the perineal wound. A small rubber tube may be attached and drawn through by the threads. A large, soft catheter, with multiple perforations having been introduced into the bladder for perineal drainage, the suprapubic incision. bladder, and abdominal wall are closed completely, except where the threads protrude, the anterior bladder wall being anchored near the recti muscles. In 9 of 11 cases the wound entirely healed within two weeks. In the remaining 2, oozing blood was allowed to block the perineal opening, making it necessary to reopen the wound for the insertion of a larger drain. Greater care in checking oozing with formalin gelatin should prevent this mishap.

The Treatment of Fistulæ.—Mertens (*Zentralbl. f. Chir.,* 1906, xxxiii, 1345) says that the usual treatment of fistulæ, curetting away the anemic and indolent granulations with a sharp spoon and producing a fresh healing surface, has the two disadvantages that it requires narcosis and leaves a larger wound. Mertens advises the employment of dry cupping to produce a freshening and hyperemia of the granulations, and says the more active the hyperemia produced the more quickly will the fistula close. He first sets the cupping glass over the mouth of the fistula with firm pressure so as to obtain the greatest suction. The effect of the sudden forcible filling of the bloodvessels is to rupture the torpid granulations, and to produce a more or less fresh wound surface with an escape of blood from the fistula. This suction is continued for about three-quarters of an hour. The bleeding stops gradually and serum takes its place. This procedure is repeated on the next and on the third day, when it may be stopped. Healing of the fistula usually occurs in about a week. Mertens admits that these fistulæ may heal spontaneously if sufficient time is permitted, but holds that when a fistula has existed seven weeks and closes under this treatment in seven days, the result should be attributed to the treatment. If it does not show an inclination to heal in a few days from this treatment there is probably a foreign body, as a necrosed bone, at fault, and this must be removed. He says that post-traumatic and postoperative fistulæ (not specific) heal by this treatment in a short time. If one does not improve in a few days there is a clear indication for an early operation for the removal of a foreign body.

Imperforate Anus; Congenital Obliteration of the Duodenum; Clubbed Hand.—HERMAN (*Jour. de chir. et annales de la Soc. Belge de Chir.*, 1906, vi, 438) reports an interesting case in which the above conditions were present. The child died three hours after a perineal operation, by which the rectal pouch was reached and the edges of the opening in the bowel were sutured to the edges of the perineal wound. The autopsy revealed an enormous distention of the stomach, which at first sight seemed to be double. The lower portion of the distended mass, however, was made up of duodenum; but both portions were filled with meconium. There was atresia of the small intestine, cecum, ascending and a part of the transverse colon; although their general conformation was normal. The appendix, of normal length, contained only a little meconium; the small intestine, cecum, and part of the colon were absolutely empty. The duodenum, beyond the dilatation of the portion already referred to, did not appear to be permeable. A part of the transverse, the descending, and the sigmoid portions of the colon contained a rather large quantity of meconium. At the end of the dilatation of the duodenum there was a true valve preventing the passage of liquids into the intestine. This case shows how the success of an operation for imperforate anus may be compromised by the existence of other anomalies incompatible with existence. Hobson, Champneys, Bundeau, and Kirmisson have reported similar cases.

The Omentum and its Functions.—DICKINSON (*Annals of Surgery*, 1906, xliv, 652) says that the numerous bloodvessels and lax tissues of the omentum allow of storage of blood when the general tension is high. By venous anastomosis through adhesions, local congestion may be relieved. Through its large surface freely exposed to surrounding parts in motion, the omentum is a rapid absorber of fluids by the blood stream. By the lymph stream it is a free carrier of white blood corpuscles, encapsulating solid particles. Through its cohesive tendency apertures in the abdomen into which the omentum has been forced by intra-abdominal pressure become more or less completely closed. Through its ready tendency to lymph formation and local proliferation, it becomes attached to infected parts, which are walled off, subsequently to be absorbed by phagocytic action, the peritoneal cavity being thereby protected. The majority of phagocytes extruded into the peritoneum for its protection come through the omentum largely from the general circulation, but in part from the tissues therein existing; subsequently to be attached to the surface of this tissue, taken into the lymph stream, and subjected to the cytolytic influences existing there. Lack of development of the omentum, or loss through operation, renders one less resistant to peritoneal invasion. Hemolymph glands of the splenic type existing in its base supplement the spleen if the latter be removed or its functions interfered with.

Vasovesiculectomy in Cases of Genital Tuberculosis.—Baudet and Kindirdjy (*Revue de Chir.*, 1906, xxvi, 749) believe that this operation is especially necessary: when there is a urinary fistula, produced and maintained by the vesicular tuberculosis; when there is rectal obstruction; when the urinary disturbances are determined by this vesiculitis and are not dependent on a cystitis; when the patients for some time under observation and treatment, medical or a previous castration, have vesicles that continue to increase in size; finally, when there exist well-marked lesions, distinctly appreciable, along the whole length of the deferential canal. Moreover, vesiculectomy appears to be the operation of choice, when one or both vesicles are very large. When the lesions of the deferential canal and of the seminal vesicles are not marked the writers do not advise vasovesiculectomy. In such a condition, according to Roux, this operation should be done to prevent the later necessity for epididymectomies and castrations, to prevent tuberculous infection of the other testicle, and to prevent a general tuberculosis. In the opinion of the authors these indications are purely theoretical, which the operation does not always fulfil. These cases appear to be especially amenable to the more economic operations on the testicle and epididymis and to medical treatment. In spite of this restriction the operative indications are very numerous and precise. There is already a strong tendency toward the operation in England, America, Switzerland, and France. Its use will undoubtedly be somewhat restrained, but Baudet and Kindirdjy are convinced that it will become classic, as has castration, in spite of the condemnation of Velpeau, and since then epididymectomies.

Operative Indications in Old Traumatisms of the Head.—Tixier (*Revue de Chir.*, 1906, xxvi, 861) says that the clinical manifestations consecutive to old injuries of the head are dependent upon a great variety of anatomical lesions, such as hypertrophy or splintering of the bone, epidural effusions of blood or serum, depressions, thickenings, or adhesions of the meninges, as well as changes in the cerebral substance. Microscopic alterations in the nervous substance also play a manifest role, as is shown by the experimental and clinical evidence. The work of Horsley has shown that excessive pressure from cerebrospinal fluid may produce exaggerated cerebrospinal excitation. Withdrawal of the fluid was followed in most cases by a disappearance or attenuation of the symptoms. Tixier thinks that an excess of cerebrospinal fluid ought to be considered in some of the more important anatomical lesions. In the case of an injury to the cranium, judged unimportant because of the lack of superficial evidence of an important lesion, but which later develops a series of complications, it is well to take into account the possibility of a previous injury to the cranium. The symptomatic indications of old traumatism are precise when the signs of localization are directly in accord with the relicts of an old fracture or contusion. The signs of Jacksonian epilepsy are the signal symptoms. Headache resisting all palliative treatment may acquire such an intensity as to make it an operative indication of the first order. There do not exist any absolute surgical contraindications. Lumbar puncture, in all cases with symptoms consecutive to old cranial injury, has an incontestible diagnostic value. It can also attenuate or cause to disappear the cerebral symptoms depending upon an excess of intracranial tension, such as the vertigoes, headache, œdema of the papilla, epileptiform excesses. This operation in some cases may be followed by a cure. In the case of recurrence or of failure, complete or relative, one should not hesitate to perform a second operation. Successive operations have great importance, since the observations of patients definitely cured only after the third operation are not rare in the literature.

The Radiotherapy of Hypertrophied Prostate.—TANSART and FLEIG (*Annales des maladies des organes genito-urinaires*, 1906, xxiv, 1841) believe that in hypertrophy of the prostate distinct atrophy results from radiotherapy. Since the perineal region, which is the better for exposure to the rays, is a sensitive part, the exposure should be prudently conducted. The strength should never exceed five units of Holzknecht, the rays marking seven on the radiochronometer of Benoist. Tansart and Fleig prefer prolonged exposures, at intervals of fifteen to twenty days, with rays of moderate intensity, rather than heavy exposures at short intervals. They continue the treatment until the complete disappearance of all physical and functional troubles. Radiotherapy is particularly indicated in: prostatics who have not attained the period of retention; young prostatics; those who, having attained retention, can only urinate with the catheter, and are not obliged—from their social position—to obtain a rapid cure; those with infected bladder; and those with renal lesions.

The Technique of Operations on the Head and Neck.—CRILE (*Annals of Surgery*, 1906, xliv, 842) restricts himself to a consideration of the control of hemorrhage, and the plan and extent of dissection in malignant tumors. Four distinct methods are considered in connection with arterial hemorrhage. The head-up posture by diminishing the arterial flow is helpful, but with the patient under full anesthesia is attended with the danger of sudden and not easily controlled cerebral anemia, because of the circulation not being sufficiently under control. If full surgical anesthesia is not employed, precise dissection is hampered. The literature shows between 2 and 3 per cent. mortality from cerebral anemia, due to ligation of the external carotid artery; while the exposure and tying of the vessel requires dangerous handling of malignant tissue. The laborious task of picking up a large number of vessels from point to point by artery forceps requires an increased length of time and becomes a decided factor in the production of shock. The fourth method includes the temporary closure of the common carotid, in the head-up position, the catching of each vessel with forceps as it is cut, and the application of the pneumatic suit to prevent cerebral anemia. Air embolism is another distinct danger from the head-up position, but is avoided by accurate dissection, which is greatly favored by the clearer field afforded by the posture and arterial occlusion. Of 18 cases of excision of the Gasserian ganglion, in the last 8 the factor of hemorrhage from the standpoint of its constitutional effect, and almost equally so from the standpoint of the obscuring of the field of operation, has been literally obviated. Great advantage is experienced by this method in operations on cleft palate, excision of the tongue or of the tonsil for malignant disease, anesthesia being carried out by tubage of the pharynx, the method of performing which Crile has described in a previous paper. In the earliest stage of malignant disease local excision may be sufficient; but it is safer always to remove the lymphatic-bearing tissue immediately draining the focus. If the lymph glands are once involved, the whole group of glands and gland-bearing tissue of that side of the neck must be sacrificed, and the key to the situation is the internal jugular vein, which is readily compensated for, physiologically. This paper is based upon experimental researches which have been heretofore published, and a clinical experience in 734 operations on the head and neck.

THERAPEUTICS.

UNDER THE CHARGE OF

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The Influence of the Roentgen Rays upon the Blood and the Blood-making Organs.—Barjon (*Médecine moderne*, 1906, No. 31, p. 245) considers that the *x*-rays are potent only in the chronic type of leukemia; they are useless in the acute and subacute types of the disease. In favorable instances their effects are marked; all the symptoms and physical signs diminish and finally disappear. Unfortunately the cure is often merely temporary, for recurrences are frequent. In other patients the improvement is limited to certain symptoms; the splenic enlargement persists, the polynuclear leukocytes do not return to the normal number; the arrest of the disease may last for several months; but the time comes when, in spite of the improvement, death occurs, the evolution of the morbid process having continued even though the essential symptoms have disappeared. The prognosis depends upon the patient's general condition, the hemoglobin percentage, and the temperature. It is essential that the number of red cells should be kept as nearly normal as possible. The technique of the application of the rays is as follows: They are applied chiefly to the splenic region; but the sternum, the extremities of the long bones, and the lymph glands are not to be neglected. The exposures should be in series intermitted by periods of rest. The patients so far treated have not been followed sufficiently long for it to be possible to make definite statements as to the efficiency of the treatment; but we are able to assert that no other means affords results in any way comparable. The operator should always be skilled both in radiotherapy and hematology.

The Roentgen Rays in Chronic Bronchitis.—T. Schilling (*Semaine médicale,* 1906, No. 38, p. 451) has noticed in a patient affected with bronchitis who had been submitted to the *x*-rays a complete cessation of expectoration during the evening of the day of the exposure. The other symptoms also were ameliorated, and after about five weeks the dyspnœa had wholly disappeared and the patient expectorated less in a week than he had previously raised in a day. In a number of others systematic employment of the rays led to the following results: Seven bronchitic patients were treated, and it was found that exposures of from six to fifteen minutes sufficed to produce the desired result. Both sides of the chest were exposed, save when the bronchitis was unilateral. Schilling is able to state that in chronic bronchitis the good effects of radiotherapy are almost immediately evident, and persist for as long as two or three months in some patients.

Electricity in Circulatory Disorders.—L. Raab (*Münch. med. Woch.,* 1906, Nos. 29 and 30, pp. 1412 and 1440) has employed electric baths in various circulatory affections and has studied their influence upon physical signs and symptoms. Baths of the galvanic current exercise a distinct effect upon the action of the heart and cause the symptoms of fatigue to disappear rapidly. To this effect is added a functional modification of the vascular system, increasing arterial pressure to its maximum. There are, however, conditions aside from acute febrile diseases and arterial lesions, in which electricity fails completely. Bad effects following the baths are rare; but in hysterical patients, tremors and pain may result from the action of the current. Elevated blood pressure, in Raab's opinion, should preclude the exclusive use of the alternating current, and he states that the itching that this current may cause when the alternations are slow (5000) may be avoided if the interruptions are increased (14,000). He considers the electric bath superior to the tepid bath in patients who react poorly to cold, and in certain respects it is superior to carbon dioxide gas baths, although these latter possess certain distinct advantages.

Serum Therapy in Anthrax and Plague.—N. D'Este Emery (*The Practitioner,* 1906, lxxvii, 597) states that several sera are now obtained which appear to be of decided value in the treatment of anthrax. Of these the best known is Sclavo's, which is obtained from the sheep or ass. The process consists in first immunizing the animals by Pasteur's method of the two vaccines, and following this by repeated large injections of virulent cultures mixed with gelatin, which seems to obviate abscess formation. The serum has been largely used on the Continent and several cures have been reported in England. The initial dose is 5 to 10 drams repeated in twenty-four hours if necessary, or four or five

5 dram doses may be employed. Sobernheim's method of immunization is somewhat different and consists in injecting the animal with a mixture of bacilli and anthrax serum. This method is supposed to give the rapid immunization of the passive method and at the same time the more lasting immunity obtained by vaccination. The serum has been mainly used in animals, but extremely good results have been obtained in man; the usual dose is 5 drams and intravenous injection is advisable. Both Selavo's serum and Sobernheim's method may be followed by rise in temperature and sweating, after which improvement takes place within twenty-four hours. The serum treatment of plague, according to the latest reports from India, is not affording the good results which might be hoped. The best serum appears to be Terni's, which has given a small reduction in case mortality, a relief of symptoms, and a lengthening duration of life in fatal cases. Duprat has treated, with the Roux-Yersin serum, twenty-nine patients with only four deaths; the doses were large—6 to 9 drams; when smaller amounts of serum were used the results were less satisfactory. Hill has found the Yersin serum valuable in the recent Natal epidemic. Most observers advise large doses and Calmette advises intravenous injection.

Galvanism in Exophthalmic Goitre.—A. D. Rockwell (*New York Med. Jour.*, 1906, lxxxiv, p. 873) considers that while much has been accomplished by surgery and by antitoxin treatment in this intractable affection, the results from galvanic treatment are quite as good, if not better, than those obtained by other means. The disadvantage of electricity is its slow action; on the other hand, it is not dangerous and might profitably be employed in connection with antitoxin. The dose must be massive, that is to say, the current should be carried up to the limit of endurance, but not high enough to injure the skin.

Methylthionine Hydrochloride in Inoperable Cancer.—Jacobi (*Jour. Amer. Med. Assoc.*, 1906, xlvii, 1545) states that this drug has rendered him much good service in from 120 to 150 cases during the last fifteen years. He prescribes the agent in pills, in small doses, 2 grains a day slowly increased to 6 grains. He considers that larger dosage is not required. The pills are made up with belladonna extract, $\frac{3}{4}$ grain daily, to relieve any dysuria which may occur. Frequently arsenic trioxide, $\frac{1}{40}$ to $\frac{1}{20}$ grain, three times daily, is added to the pill; strychnine or nux vomica extract may also be added if indicated. While Jacobi makes no claim that methylthionine hydrochloride possesses any curative action, he feels sure that in instances of inoperable cancer it will do a great deal of good and that by its use many patients have been kept alive and even in a condition to work for from two to eight years longer than would otherwise have been the case.

The Treatment of Chlorosis.—H. Rosin (*Die Therapie der Gegenwart*, 1906, No. 7, p. 298) advises in this affection baths at 104° F., three times weekly, at first of ten minutes' duration, later gradually increased to twenty minutes. The same temperature should be maintained throughout the procedure; an ice-cap should be kept upon the patient's head and at the end of the bath she receives a cold douche lasting a few seconds. She is then dried with a towel and allowed to rest for an hour. At first the baths cause an unpleasant nervousness, which disappears after three or four applications of the treatment; at this time a general improvement is noted, and recovery is said to be evident by the time twelve baths have been taken. If the chlorosis is a manifestation of organic disease, such as tuberculosis or nephritis, the treatment naturally is ineffective. In instances of true chlorosis, recurrences which may take place should be treated in the same manner as the original attack.

Medicinal Treatment of Gastric Displacements.—Fenwick (*Edinburgh Med. Jour.*, 1906, xx, 395) states that in uncomplicated instances drugs are seldom of much value and the treatment is chiefly symptomatic. For anorexia, nitrohydrochloric or phosphoric acid, combined with a bitter infusion, may be given between meals. If extreme weakness is present, tonics such as strychnine, nux vomica, or cinchona may be prescribed; in neurasthenic patients, cod-liver oil, the compound syrup of hypophosphites, elixir of phosphorus, or sodium formate may prove beneficial. Pain after eating and flatulence may be relieved by compound bismuth mixture, with or without morphine. Preparations of pancreatin, pepsin, or peptenzyme may aid the process of digestion. The presence of myasthenia requires the addition of phenol to the bismuth mixture, and in instances complicated by colitis full doses of bismuth salicylate, cyllin, or guaiacol should be employed. The selection of an aperient is important. As a rule, severe purgatives are to be avoided and reliance is to be placed in small doses of cascara and euonymin combined, if necessary, with belladonna and rhubarb; a confection of guaiacum, senna, and ginger may be employed to advantage. If the colitis is associated with constipation nothing is so effectual as a small dose of castor oil each morning before breakfast, but if severe neurasthenia exists purgatives must be omitted and a daily movement must be secured by soapsuds enemas or glycerin injections. Carlsbad and other salts are indicated in myasthenia with stagnation of food, since they effect a form of internal lavage, sweeping the fermenting gastric contents into the intestine. In other respects gastroptosis is to be treated, as far as medicines are concerned, identically with chronic gastritis and gastric atony.

The Treatment of Rectal Constipation.—According to H. Strauss (*Therap. Monats.*, 1906, No. 8, p. 373) stasis of the feces in the rectal ampulla is responsible for 10 per cent. of the instances of constipation. It is the result of a loss of the reflex which causes the expulsion of the fecal masses when these have reached the rectum, and this is chiefly caused by a disinclination on the part of the patient to go to stool, perhaps on account of the pain which is induced by the act, as in gonorrhœal prostatitis, uterine disease, etc. Treatment consists of local measures and in regular visitation of the stool, even when inclination is not present, at a certain time each day. Faradism, vibratory massage, and cold irrigations are useful adjuvants. In addition, the employment of a specially constructed syringe, by means of which three ounces of oil may be injected into the rectum, may be prescribed. When the scybalæ are so hard that they provoke a spasm of the sphincter when an attempt at passage is made, the patient may be advised to attempt defecation in a vessel containing steaming hot water.

Strontium Lactate in Hemophilia.—J. B. NIAS (*Semaine médicale*, 1906, No. 36, p. 428) has obtained an excellent result in a hemophilic patient in whom calcium lactate and the administration of thymus gland had proved conspicuous failures, by prescribing strontium lactate. The dose given was 15 grains, which amount reduced in about an hour the coagulation time of the blood by a third. Double dosage produced no better effect. The result was not permanent and the patient was furnished with powders of the drug to be taken whenever hemorrhage occurred. By this means the patient's bleeding could be stopped within twelve hours when previously it would persist for several days.

The Roentgen Rays in Neuritis.—J. H. CUMROE, (*New York Med. Jour.*, 1906, lxxxiv, 740) reports six instances of neuritis in which radiotherapy has been employed, and from them draws the following preliminary conclusions: (1) The *x*-rays should be employed in neuritis only after all other judicious methods of therapy have failed. (2) The exposure should be directed over as wide an area as may be safe, and, at first, exposures should be frequently repeated. (3) The strength of the current and the duration of the exposures should, as a rule, be in direct ratio to the approximate distance of the affected nerve structures from the overlying skin. Idiosyncrasies should be carefully sought. (4) Relief, if obtained, usually makes its appearance early in the treatment. (5) Obstinate cases should receive "tonic" treatment with a medium tube for some time after apparent cure, so as to assure permanency. (6) Postoperative treatment in nerve resections is highly recommended.

The Treatment of Simple Insomnia.—LEMOINE (*Jour. de méd. de Paris*, 1906, No. 39, 423) advises patients affected with this condition to take at evening a bath to which an infusion of linden leaves has been added. This should be of about body temperature and of a duration of three-quarters of an hour. The bath is followed by a light meal without beer, wine, or coffee. Three hours later the patient retires after taking a cup of hot infusion of linden leaves to which about two teaspoonfuls of the following mixture has been added: Potassium bromide, 5 parts; hydrated chloral, 10 parts; syrup of lactucarium, 20 parts; cherry-laurel water, 10 parts; lettuce-water, 120 parts; for this may be substituted 12 grains of sulphonal combined with 4 grains of sodium bicarbonate, or 3 grains of chloralose. These doses may be repeated later if necessary. If the insomnia is associated with nervousness or anxiety heroin may be given as follows: Heroin hydrochloride, 0.003 to 0.008 parts; cherry-laurel water, 10 parts; linden-water, 50 parts. Dose two teaspoonfuls every hour. If there is danger of the patient becoming habituated to the drug paraldehyde should be substituted.

PEDIATRICS.

UNDER THE CHARGE OF

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The Routine Treatment of Scarlet Fever with Chloral Hydrate.—B. Franklin Royer (*Penna. Med. Jour.*, January, 1907, p. 286) describes the results of a study of the kidney condition of 800 cases of scarlet fever, treated with routine doses of chloral hydrate, as compared with the renal conditions observed in 756 cases having the usual remedies. The study was made in the wards of the Philadelphia Municipal Hospital, beginning in 1904, with the object of determining the value of chloral hydrate, which was advocated in 1896 by Dr. James C. Wilson. Dr. Wilson believed that he had secured an increased diuresis thirty-six to forty-eight hours after the administration of chloral had been begun. He thought that in some way chloral protects the functionating part of the kidney tissues, that the nervous symptoms were greatly modified, that the itching was allayed, and that the patient was made more comfortable. A few weeks' trial convinced the hospital staff that Dr. Wilson's claims were for the most part easily proved and that chloral hydrate did appear to be the most useful remedy yet advocated in the treatment of scarlatina for making the patient more comfortable, ameliorating the nervous symptoms, and controlling the itching. The effect upon the circulation was next carefully studied. After treating nearly 1000 patients with doses of sufficient size to produce light somnolence, it was decided that chloral did not materially depress the circulation, and in no case did the drug act clinically as a serious depressant. At first the drug was given to patients greatly prostrated on admission and discontinued after a few doses were given because of prejudice against it. After a larger experience it was rarely discontinued, no matter what the degree of depression might be. One claim was not confirmed, viz., that of early diuresis. An increased flow of urine twenty-four to forty-eight hours after admission undoubtedly does occur, but the same increase was noted in patients treated by other remedies. This Royer attributes to better nursing, the systematic use of water in definite quantities, insisted upon whether the patient wants it or not, and the greater ease with which liquids are swallowed as the angina subsides.

The special study to which this paper is devoted relates to the kidney condition met with in the postfebrile period: 800 cases were treated with routine doses of chloral in sufficient quantities to produce light somnolence, and continued in the same dose for five or six days after the fever subsided. A series of 756 patients treated by the usual remedies (a few of them perhaps having a dose of chloral to quiet extreme restlessness at night, most of them having no chloral at any time) was analyzed in exactly the same way. All histories were excluded in both series in which other infectious disease or other serious illness was combined with scarlatina; 3 patients died in the chloral group, and 4 in the usual remedy group. In the febrile period no material difference was found in the number of those having either mild, transient, or severe nephritis. In the postfebrile period the percentage of those having postfebrile albuminuria was 4.25 per cent. in the chloral cases as against 5.68 per cent. in the series treated without chloral; a transient postfebrile nephritis occurred in 3.5 per cent. as against 5.29 per cent. without chloral; and severe postfebrile nephritis occurred in 2 per cent. as against 2.38 per cent. without chloral. The total of all forms of postfebrile nephritis was 5.5 per cent. in those having chloral as against 7.76 per cent., or an apparent total saving of 2.17 per cent. of postfebrile nephritis. A separate classification of patients to whom antitoxin had been given apparently demonstrates that nephritis is a little more frequent in those patients having antitoxin. Another table shows the day of the disease when nephritis may be expected. The greatest danger is shown to be the period from the twentieth to the thirty-second day, or the third week and the early part of the fourth week. Royer concludes that this study would seem to justify the more extended use of chloral in the treatment of scarlet fever, and a more detailed study as to how it acts on the kidney itself.

Lobar Pneumonia as a Complication of Diphtheria.—The occurrence of lobar pneumonia in the course of diphtheria has always been regarded as an exceptional event. The earlier writers, such as Barthez and Rilliet and Cadet de Gassicourt, stated that the pneumonia which complicated diphtheria was always lobular in character. Sanné is the only authority who has held that this condition is other than a rarity. He has found it in 48, or 3.2 per cent., of his 1500 cases of diphtheria. most of these occurring in laryngeal cases, and only 4 recovered, Recent writers are agreed as to the rarity of secondary lobar pneumonia in diphtheria. J. D. ROLLESTON (*Brit. Jour. Children's Diseases*, December, 1906, p. 536) presents a paper based upon his observations made on 1000 consecutive cases of diphtheria under his care at the Grove Hospital (London) during the past four years. Lobar pneumonia occurred in only 7 cases (0.7 per cent.). All the patients were children, their ages ranging from about two years to seven years. As 266 of the total number were above the age of seven years, childhood appears to be a predisposing factor. Three of the patients were males, 4 females. Only 3 were cases of laryngeal diphtheria, 2 requiring tracheotomy; of the remainder 3 were of a severe faucial type, and 1, the most typical, was a mild faucial case. It is noteworthy that in 15 cases of bronchopneumonia that occurred in the 1000 cases, 9, or 60 percent. were found in laryngeal cases, 7 of which were tracheotomized. With one exception, occurring in the ninth week, all the cases arose within the first ten days of the disease; in 3 cases the pneumonia developing before the throat became clean; in 2 the membrane disappeared from the throat on the same day the pneumonia began. In the remaining case the throat had already been clean six days.

In 3 the onset was abrupt, occurring after the subsidence of the initial pyrexia; in the remaining 4 the initial pyrexia had not yet subsided, but was increased by the supervention of the pulmonary condition, The pneumonic process showed a predilection for the right lung, especially for the lower lobe. In 5 the right lower lobe alone was affected, in 1 the lower lobes of both lungs, and in 1 the right upper lobe alone.

Of interest in the diagnosis is the fact that the initial pyrexia of diphtheria is usually of short duration, and tends rapidly to fall after the injection of antitoxin. A sudden rise or an exacerbation of the initial pyrexia should therefore draw special attention to the condition of the lungs. According to v. Jürgensen the diagnosis of lobar pneumonia can only be settled by the absence of a widespread capillary bronchitis.

The more favorable prognosis of lobar pneumonia as compared with bronchopneumonia is illustrated in Rolleston's cases by the fact that whereas none of the 7 cases proved fatal, only 3 of the 15 cases of bronchopneumonia among the 1000 cases of diphtheria recovered—a mortality of 80 per cent.

The rarity of lobar pneumonia in diphtheria suggests that its occurrence is but a fortuitous one, Baginsky in particular regarding it as entirely accidental. Against this view should be weighed its predilection for children and its occurrence at an early stage of the disease. In addition to the characteristics already noted it may be stated that such cases resemble the primary lobar pneumonia of children in being atypical in the following respects: Absence of expectoration and of marked respiratory trouble, an occasionally remittent or even intermittent pyrexia, the frequent occurrence of lysis, its relative benignity, and absence of any sequels except empyema.

A Large Retroperitoneal Dermoid Cyst in an Infant.—RAYMOND JOHNSON (*Brit. Jour. Children's Diseases*, December, 1906, p. 549) reports a case of a child, aged two years and nine months, with a large, elastic tumor in the left half of the abdomen, presenting characteristics similar to those met in a sarcoma of the kidney. The lumbar spine showed a well-marked lateral curvature to the right, with rotation of the vertebræ, and in the left loin were two small fluctuating swellings not apparently connected with the tumor in the abdomen. Before operation the case was regarded as probably tuberculous disease of the spine, with lumbar and large retroperitoneal chronic abscesses. An exploratory incision in the loin revealed the true nature of the case. Part of a small cyst removed from the posterior part of the tumor presented the naked eye and microscopic characters of a dermoid tumor.

OBSTETRICS.

UNDER THE CHARGE OF

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Induction of Premature Labor and Accouchement Force.—WILLIAMS (*Surg., Gyn., and Obst.*, September, 1906) in 5000 labors in the obstetrical department in the Johns Hopkins Hospital found it necessary to induce premature labor in 11, and to practise accouchement forcé in 100 cases. Labor was induced by the introduction of a bougie for cardiac lesions, pre-eclamptic toxemia, retention of dead fœtus, hydramnios, infection, and overdevelopment of the child. All the patients recovered. The cervix was uninjured in 9, and slightly torn in 2 cases. In 83 cases the cervix was dilated manually by Harris' method. This was done for eclampsia in 33 cases, toxemia in 7, placenta prævia in 12, and danger to mother or child during labor in 31. In eclampsia 2 patients died as the direct result of the operation, 1 from infection, and 1 from incomplete rupture of the uterus. In these 33 cases the cervix was uninjured in 11, slightly torn in 3, and deeply torn in 19; and in 6 of the latter the laceration was so deep as to give rise to serious hemorrhage and require immediate repair. In 7 cases of toxemia threatening eclampsia, it was effected by manual dilatation, with 2 deaths, 1 from toxemia, and 1 from incomplete rupture of the uterus, due to the operation. In placenta prævia the same operation was performed in 12 cases; and 1 patient died from hemor hage following incomplete rupture of the uterus caused by the operation. In 31 patients delivered by manual dilatation, because of some danger arising during labor, 1 maternal death occurred, caused by partial intestinal obstruction and thrombosis of the vessels of the left leg, originating during pregnancy. The mortality rate of the operation was 4.82 per cent. The cervix was deeply lacerated in 39.71 per cent. In 17 per cent. it was so badly torn as to cause profuse hemorrhage, and require immediate repair. The results were much worse in placenta prævia than in any other condition. In 15 cases dilatation was effected by the use of de Ribes' bag. All of the mothers recovered, and the cervix was deeply torn in 6 cases. In 2 of these, hemorrhage was so great as to require immediate repair. There were 2 cases of vaginal Cesarean section, one for eclampsia, and one for toxemia with twin pregnancy. One of these patients died, partly from hemorrhage and partly from intoxication. Technically, the operation was successful. In reviewing these methods, the use of the bougie is evidently safest, but it is uncertain and tedious in many cases. Manual dilatation is difficult and dangerous when the cervix is intact and the internal os not obliterated. If the cervical canal is obliterated and the external os offers little resistance, manual dilatation is comparatively safe. In placenta prævia manual dilatation is extremely dangerous, as deep tears occur in 50 per cent. of cases, and most of these were complicated by hemorrhage and required suture. The use of the dilating bag seems indicated in placenta previa when haste is not essential, and when the cervix is not extremely rigid. Vaginal Cesarean section is not adapted for general use in private practice and requires special surgical training. Very large specula should be employed, and usually both anterior and posterior incisions may be made. The uterus should be packed after delivery to guard against relaxation. In obstetrical hospitals this operation will be found successful. In general, the induction of premature labor was not adopted for moderate pelvic contraction. Such patients were allowed to go to term, and labor was terminated by forceps, pubiotomy, or Cesarean section, as indicated in the individual case. When haste is not imperative, the method of the bougie may be employed in all other conditions. In eclampsia, if the circumstances are favorable, vaginal Cesarean section should be useful when the cervix is undilated and resistant. When the cervix is obliterated and the external os only remains, manual dilatation is indicated. The Bossi dilator was not employed, and does not seem indicated. Vaginal Cesarean section is safer when the cervix is not softened, and when the cervix is obliterated manual dilatation may be performed. In placenta prævia the ideal method of treatment seems to lie in the use of the dilating bag, or bipolar version, with very gradual extraction. When danger arises to mother or child during labor, the operator should hesitate to have recourse to accouchement forcé. The dangers of this procedure may be greater than the danger which threatens the mother.

Eclampsia Four Weeks after the Birth of the Child.—Vaquez (*Zentralbl. f. Gyn.*, 1906, No. 41) reports the case of a patient who, four weeks after a normal labor, came to the hospital complaining of weakness, headache, and slight œdema of the legs. The patient's mother had had eclampsia. On examination, the pulse tension was high, and the heart sounds strongly accentuated. The urine contained a considerable percentage of albumin. In the twenty-four hours following, the patient had 17 typical eclamptic convulsions, the temperature rising to 103.5° F. After removing considerable blood from the arm, the convulsions ceased and did not recur. The blood pressure slowly grew less and the secretion of urine increased. After the fourth day from the begining of the eclampsia the patient rapidly improved.

Criteria of Full-term Birth.—Holzbach (*Monats. f. Geb. u. Gyn.*, 1906, xxiv, Heft 4) contributes a paper in which he describes those signs from which the full term development of a newborn child may be estimated. In reviewing the literature on the subject, he finds that the length of gestation has been considered by some important in estimating the full term development of the child. As gestation differs in different patients, it is impossible to form a definite opinion concerning the length of a gestation which may produce a fully developed child. Regarding the length of the child, Issmer has called attention to the fact that 61.8 per cent. of fully developed children in a considerable number, varied in length from 48 cm. to 50. In 31 premature children, Holzbach found three 48½ cm. long. We cannot assert that a child 50 cm. long is more completely developed than one 48 cm. long. The weight of full-term children differs so greatly that this cannot be taken as an absolute criterion. If the attempt is made to take the development of the finger-nails, the presence of lanugo, or the condition of the genital organs as indicating complete development, we find here that no one of these is absolutely reliable. In 30 premature children, Holzbach found 18 whose finger-nails were well developed and firm. The cartilages of the ear and nose were well-developed in 42 per cent. In 17 per cent. of premature children lanugo was not present. This statement differs essentially from that of Herz, who, in 96 premature children, found lanugo on the face, the buttocks, and extremities in 1 each. The presence of comedones was observed in premature children in the majority of cases. As regards the condition of the genital organs, the labia were found fully developed in 48 per cent. of premature female children. In 72 per cent. of male children the testicles were already in the canal or in the scrotum. The greater fontanelle varies so much in size that its condition cannot be taken as a means of determining the complete development of the infant. So far as the separation of the umbilical cord is concerned, the stump of the cord loosens earlier in premature children than in those at full term. Some deny this observation, and believe that no difference can be demonstrated between the two. As regards the weight of the placenta, it is unquestionably greater in full-term children than in the premature. Variations in weight of 300 or 400 grams between the placentæ of fully developed children were also observed.

Examination of the fœtal skeleton is also not conclusive, as children vary greatly in the degree of ossification at full term. The weight of the various fœtal organs, and the proportionate development of the child, are of assistance in estimating full development. Holzbach considers of especial value the relation between the circumference of the shoulders and the circumference of the fetal cranium. It is evident from this and other papers upon the subject that a child cannot be declared as fully developed because it possesses one of the conditions present at full term. It must show a considerable number of the various criteria of full development.

Pubiotomy and Induced Labor.—FEHLING (*Monats. f. Geb. u. Gyn.*, 1906, xxiv, Heft 4), from the statistics of the Strassburg clinic, believes that premature birth does not jeopardize the existence of the child after the first few weeks. Pubiotomy cannot be considered a substitute for induced labor. It should be selected or primiparæ with contracted pelves as a substitute for craniotomy; also for multiparæ who decline Cesarean section; and when complications arise in labor because of disproportion. Induced labor is especially successful among married patients who have lost one or two children in difficult birth.

The Indications for the Management of Labor in Contracted Pelvis.—PFANNESTIEL (*Monats. f. Geb. u. Gyn.*, 1906, xxiv, Heft 4) urges increased practise and adoption of induced labor in contrast with pubiotomy. and Cesarean section. When the pelvis is highly contracted, a woman should be sent to a hospital for treatment. In all these cases the effort should be to allow a natural attempt at parturition, giving the patient every assistance to complete the birth spontaneously. The forceps can seldom be used to advantage, especially in flat pelves. Version and extraction are dangerous in highly contracted pelves of symmetrical shape When the pelvic contraction is slight, the prognosis is slightly better, and is best in cases of simple flat pelves. In multiparæ with simple flat pelves prophylactic version gives good results. In primiparæ when the true conjugate is from 7 to 8.5 cm., induced labor is usually successful. When the pelvis is flattened only induced labor, with version, is useful. The use of the elastic dilating bag is also indicated in these cases. When the pelvis is symmetrically contracted, it is well to avoid the use of bags, and if necessary, to employ a bougie instead. When the pelvis is highly contracted, induced labor must be declined. If the child's life is to be saved, and the soft parts permit rapid dilatation, pubiotomy may be performed, but otherwise Cesarean section should be chosen.

GYNECOLOGY.

UNDER THE CHARGE OF

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Hemorrhage following Vaginal Hysterectomy.—MENDES DE LEON (*Zentralbl. f. Gyn.*, 1906, No. 40) reports a case in which clamps were used, which were removed at the end of twenty-four hours. On the seventh day such a profuse hemorrhage occurred that the patient became almost pulseless, but it was controlled by tamponade and she afterward made a good recovery. De Leon stated that this was the first case in which he had observed hemorrhage after the clamp-operation and attributed it to the opening of a large artery following the usual sloughing process. This was less likely to occur, he thought, when ligatures were used. In the discussion of this subject before the Dutch Gynecological Society, Stratz said that he had abandoned the use of clamps in vaginal hysterectomy because the patient had more pain than when ligatures were applied, the operation was less clear and exact, and there was more danger of secondary hemorhage. He was accustomed to closesthe peritoneum. He generally used from twelve to fourteen ligature , but the operation seldom consumed more than five or ten minutes more than the clamp operation.

Intra-abdominal Shortening of the Round Ligaments.—SPERLING (*Ztsch. f. Gyn.*, November 7, 1906, p. 206) commends this operation highly for the following reasons: (1) Freedom from danger. (2) Infrequency of recurrence, especially after parturition. (3) Avoidance of the risk of interruption of pregnancy. (4) The eliminating of subsequent dysmenorrhœa. (5) Avoidance of adhesions and consequent intestinal obstruction. (6) The danger of hernia is lessened by employing the transverse incision. (7) Adhesions overlooked during the examination are discovered and broken up. (8) Subsequent conception is rendered more probable. (9) There is less danger of displacing the tubes and thus forming the occurrence of ectopic gestation. (10) The uterus is more likely to be retained in its normal position in the pelvis. In general Sterling prefers intraperitoneal shortening of the ligaments to vaginal fixation or Alexander's operation.

Drainage of the Peritoneal Cavity.—D'ERCHIA (*Rassegna di ost. e gin; Zentralbl. f. Gyn.*, 1906, No. 40) distinguishes peritoneal from retroperitoneal drainage, meaning by the latter term that the peritoneum is sutured over the gauge drain. He also refers to "prophylactic" and "curative" drainage. He believes that in complicated cases drainage should reduce the mortality from 20 to 3 or 4 per cent. A series of seventy-two cases is appended in which abdominal drainage with gauge was employed in eleven, and vaginal in forty-seven. (It is interesting to note the unwavering faith of the writer in a method which is fast becoming obsolete. Most surgeons now regard drainage as rather an unfortunate necessity and resort to it only when absolutely necessary. It is a question if gauze is not really used for tamponade, rather than for its capillary action—H. C. C.)

Examination of the Stomach in Pelvic Troubles.—Winkler (*Berl. klin. Woch.*, 1906, No. 33) reports forty-one cases in which he examined the stomach contents. In only 29.9 per cent. was the secreting function normal. Hence, the deduction that the gastric symptoms accompanying uterine displacements, pelvic tumors, etc., are mainly due to disturbances of the secretory and motor functions. Gastroptosis was noted in eighteen out of thirty-one cases.

Winkler concludes that hyperchlorhydria is common in connection with pelvic troubles, and that treatment directed to the latter alone has little effect on the gastric condition.

Aspirin in Gynecology.—Chidichimo (*Ann. di ost. e gin.; Zentralbl. f. Gyn.*, 1906, No. 40) reports the results of a series of experiments with dogs, in which he found that the lethal dose was about one-half grain to each two pounds of body weight. Large doses caused temporary lowering of the arterial tension and slowing of the pulse; the heart sounds remained regular except when the dose exceeded the therapeutic limit, nor was there any change in temperature or the respirations. Aspirin exercises a decided contracting action upon smooth muscles, especially those of the uterus, whether gravid or non-gravid, the effect being noted from fifteen to thirty minutes after administration of the drug. Therapeutically Chidichimo advises that from 30 to 45 grains be administered daily. He has never observed any ill effects.

Metaplastic Processes in Uterine Polyps.—Kaufmann (*Abstract of Monograph in Zentralbl. f. Gyn.*, 1906, No. 39) presents the results of his studies of serial sections of cervical polyps, the pedicles of which were covered with cylindrical epithelium, while the distal portions presented several layers of squamous cells. He believes that the latter represent a metaplastic process and eventually replace the cylindrical epithelium. Hence the inference that in so-called "combined" carcinomas of the uterus, in which both squamous and glandular epithelium are found, the former replaces the latter over certain areas.

Epithelioma of the Uterus and Vagina.—Bjorkenheim (*Abs. of Mono. in Zentralbl. f. Gyn.*, 1906, No. 39) found in the endometrium of young subjects yellow spots, which, on microscopic examination, presented the appearance of ordinary squamous epithelium, the cells having large nuclei disposed in rows parallel with the surface. The line of demarcation between the vaginal and uterine mucous membranes was thus clearly defined, though it differed in different specimens.

Lutein Cysts and Chorioepithelioma.—Burger (*Zentralbl. f. Gyn.*, 1906, No. 39) presented to the Vienna Obstetrical Society a specimen from a patient aged thirty-two years, who, after missing three menstrual periods, had passed a vesicular mole. Six weeks later she was admitted to the clinic on account of metrorrhagia and rapid enlargement of the abdomen. The diagnosis of double ovarian cystoma was made. On opening the abdomen, besides the cysts, it was found that the uterus contained an interstitial tumor, so that it was also extirpated. It contained several vesicular moles, presenting the histological structure of incipient malignant chorioepithelioma. The ovarian neoplasms were lutein cysts. Burger calls attention to the fact (noted by himself and others) that these cysts are not really derived from the corpus luteum, but from the proliferation of atresic follicles and the lutein transformation of their cells, hence, the name follicle-lutein cysts. He sees no etiological relation between these and the development of vesicular mole; on the contrary the circulatory disturbances due to the presence of the latter are more likely to lead to cystic degeneration of the ovaries. He believes that curettement in these cases is unwise, but that a radical operation should be performed at once.

Gonococcic Peritonitis.—LEOPOLD (*Zentralbl. f. Gyn.*, 1906, No. 43) reports the case of a patient, aged twenty-eight years, who on the sixth day after a normal delivery developed peritonitis. Bacteriological examination of the urethral and vaginal secretions showed numerous gonococci. On the following day laparotomy was performed, a quantity of purulent fluid being removed, which gave pure cultures of Neisser's coccus. Incision was made in both groins, with counter-openings in the loins; rubber drainage-tubes were inserted, a third drain being carried through into the vagina; all were repeatedly flushed with normal salt solution. The patient improved at once and made a good recovery. Commenting on this case, Leopold states that it was impossible to trace the time of infection, though the history and result of the bacteriological examinations left no doubt as to the origin of the peritonitis. It was particularly instructive from its bearing on obscure cases of puerperal infection. Importance is attached by him to the repeated flushings of the cavity after operation. (We had a similar unreported case, in which infection undoubtedly occurred two or three days before labor, as proved by the vaginal discharge and the fact that the child lost one eye from gonorrhœal ophthalmia. Unfortunately permission to open the peritoneal cavity was not obtained until the patient's condition was such that recovery was impossible. The case had been diagnosed as perforative appendicitis, occurring on the fifth day after delivery.—H. C. C.)

The X-rays in Menorrhagia.—GORL (*Zentralbl. f. Gyn.*, 1906, No. 40), reasoning from the reports of Tilden Brown and Halberstädter regarding the action of the *x*-rays on the ovaries and testicles in producing sterility, and the experience of Faveau de Courmelles in forty-five cases of fibromyoma, was induced to use this agent in the treatment of hemorrhage from the myomatous uterus. His success led him to infer that the atrophic action of the Röntgen rays on the ovaries was especially useful in cases of menorrhagia in which an operation was contraindicated on account of serious cardiac or renal disease. One application a week is advised.

DISEASES OF THE LARYNX AND CONTIGUOUS STRUCTURES.

UNDER THE CHARGE OF
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OF PHILADELPHIA.

Micro-organisms in the Mouths and Throats of Healthy Persons.—In a study of the nature of micro-organisms found in the mouths and throats of healthy persons J. B. RUCKER, JR. (*Univ. Penna. Med. Bull.*, October 1906) reports a series of observations on twenty individuals who considered themselves in good health. He found streptococci and diplococci in all of them. Streptococcus mucosus was found in 70 per cent.; atypical pneumococcus in 50 per cent.; and typical pneumococcus in 25 per cent. It was learned that organisms of the same species may behave differently in their action on starch if the organisms are from different sources.

Tonsillitis.—In a paper on "The Relation between Tonsillitis and Acute Articular Rheumatism with Particular Reference to the Prophylaxis of Postanginal Rheumatism" (*Amer. Med.*, September, 1906) CHARLES F. KIEFFER, U. S. A., reports unusual success from a routine treatment as follows: A full mercurial purge on admission; gargling or spraying the throat with a saturated solution of sodium bicarbonate, followed immediately by the application of finely powdered aspirin to the tonsils. These applications are made thrice daily. In the series of cases thus treated the duration of the disease was but three days and not one instance of rheumatism developed.

Tuberculous Tumor at the Vault of the Pharynx.—SCHILLING (*Verhandlungen des Vereins Suddeut. Laryng.*, 1906) reports a case of tuberculous tumor in a healthy patient, aged fifty years, with normal lungs. The growth was removed with the incandescent snare, and a recurrence half a year later was destroyed with the galvanocaustic burner in two sittings. Schilling states that he has read of but two similar cases in literature, both reported by KOSCHIER (*Wien. klin. Woch.*, 1895).

Rupture of the Œsophagus into the Pleural Cavity.—THOMAS H. POTTER (*Amer. Med.*, September, 1906) reports a case of a man, aged forty-four years, who, after partaking of a liberal amount of whisky, beer, and free lunch, was suddenly attacked with a sharp pain in the precordial area, extreme physical weakness, and unquenchable thirst. In about three hours emphysema was noted at the root of the neck, and it rapidly extended over the chest, face, and arms. Death ensued eleven hours after the onset. The autopsy revealed three small ulcers, with rupture of the œsophagus at the site of one of them. The conclusions drawn were that the cold beer had caused spasmodic contraction of the œsophagus with rupture into the pleural cavity through the thin walls at the point of ulceration.

Œsophagotomy.—In reporting two cases of œsophagotomy performed for the removal of dentures impacted in the gullet, WALKER DOWNIE (*Jour. Laryn., Rhin., and Otol.*, March, 1906) states that the gullet wall was not stitched, but the wound was lightly packed and allowed to granulate. The safety of this method, he states, cannot be overestimated. A rubber tube was passed through the nose into the stomach, by which the patient was fed, and this tube was retained in one case for three days and the other for four, and in each case the wound healed satisfactorily and completely without any complications.

Perforations of the Nasal Septum.—M. A. GOLDSTEIN (*Jour. Laryn., Rhin., and Otol.*, July, 1906) states that but little attention had been directed to perforations of the nasal septum and their most satisfactory treatment. He uses the Ballenger single tine swivel knife, and is enabled to cut away the rim of the cartilage smoothly and equidistant from the mucous edges along the whole circumference of the perforation. He uses a straight, sharp, Freer elevator in elevating the mucous membrane. The results have been uniformly successful.

The Rosenmueller Fossæ and Otitis Media.—THOMAS L. BRUNK (*Laryngoscope*, August, 1906) contends that in all cases of otitis media, due to infection, the primary cause lies in the diseased fossæ of Rosenmueller which are more or less filled with granular tissue which should be curetted. Several cases are detailed from some two hundred in the practice of Brunk to demonstrate his contention, and the favorable results from curetting the fossæ, which he accomplishes with a sterilized finger in preference to surgical instruments.

Paraffin Injections for Nasal Deformity.—At a meeting of the Laryngological Society of London, March 2, 1906, WALKER DOWNIE (*Jour. Laryn., Rhin., and Otol.*, March, 1906) reproduced a number of recent photographs of some cases of paraffin injections for nasal deformity, as well as other photographs taken immediately after treatment four years previously, which demonstrated that the shape of the nose was practically identical with photographs taken two weeks after operation, and that the injected paraffin can be felt as a well-defined firm mass, occupying the same site as it did immediately after injection. From some experiments and observations which he has made, Downie believes that the paraffin breaks up on its introduction, runs along the cellular planes, and is not eaten into later by the living tissues. It is held in position by the trabeculæ of the minute planes of cellular tissue in the same way as the fat of adipose tissue is kept in place. In his own cases he has never seen migration or wandering of the paraffin after it has once set. When paraffin has been found in other parts of the face he believes that it has reached those regions while the injections were being given, and while the paraffin was still in its fluid state. Dr. Downie uses paraffin with a melting point of 106° to 108° F. He employs the electric current to keep the needle heated so as to maintain the fluid state of the paraffin during the procedure. At present he no longer employs either local or general anesthesia.

Laryngectomy in Two Stages.—LE BEC (*Ann. des maladies de l'oreille, du larynx, du nez, et du pharynx,* Septembre, 1906) reports a sixth case of total laryngectomy in two stages. The first stage comprised section of the trachea and its attachment to the skin anteriorly, a silver cannula being placed therein. Twenty days later the entire larynx was removed and an œsophageal tube was introduced through the nose into the stomach. The pharyngeal muscles were sutured in two planes around the tube, and all the soft parts were sutured with catgut. Two drainage tubes were placed at the sides and the skin sutured vertically. The sutures were removed on the twelfth day, the drain on the sixteenth, and the tube on the twentieth. Recovery took place without any special reaction, and the whispering voice had become quite perceptible. The growth had occupied both vocal bands and did not extend beyond the larynx. It was found to be an ordinary squamous epithelioma. Le Bec insists very much upon this procedure in two stages which really reduces the traumatism to its minimum, and has the advantage of allowing the patient time to recover strength between the two operative sittings

PATHOLOGY AND BACTERIOLOGY.

UNDER THE CHARGE OF

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The Presence of Syphilitic Antibodies in the Cerebrospinal Fluid of Paretics.—Based upon the work of Bordet, Gengou, Moreschi, Neisser, and Sachs, it has been possible for Wassermann and Bruck to devise a method by which the presence of minute traces of bacterial antibodies may be detected in the body fluids. It has been shown that if small quantities of blood serum from a syphilitic patient are added to a clear saline extract of the tissues from a syphilitic fœtus, this mixture is capable of preventing the action of fresh complement-containing serum, so that if one adds to this mixture fresh serum and sensitized red blood corpuscles, hemolysis does not proceed. The syphilitic blood serum or extract of syphilitic fœtal organs alone, or a mixture of syphilitic blood serum and saline extract of non-syphilitic fœtal organs, will not prevent hemolysis. Using this method WASSERMANN and PLAUT (*Deut. med. Woch.*, 1906, xxxii, 1769) have investigated the spinal fluid of forty-one paretics. Thirty-two of these spinal fluids when treated with a saline extract of syphilitic fœtal organs inhibited completely or almost completely hemolysis of sensitized red blood corpuscles in the presence of complement-containing serum. In four cases there was only slight inhibition and in five cases none. With twenty-two control spinal fluids from non-paralytics and two cases of acute meningitis, hemolysis was complete. Wassermann and Plaut believe that the patients, whose spinal fluids when tested by this method showed the presence of antibodies, must have had syphilis. In two cases, moreover, it was possible to show that the quantity of antibodies in the spinal fluid was greater than that in the blood serum. Although the number of experiments performed so far is too small from which to draw conclusions, they would suggest that in general paralysis the continuous production of antibodies by the nervous system may in some way bring about an injury to the nerve cells.

Experimental Studies on Syphilis.—Metchnikoff and Roux (*Ann. de l'Inst. Past.*, 1906, xx, 785), in the fifth communication upon this subject, publish some interesting facts and experiments to show that the syphilitic virus, after several passages through monkeys, becomes attenuated in its virulence. Throughout their previous work Metchnikoff and Roux have endeavored to find some way of accomplishing this result. In 1904 they suggested that Macacus rhesus, since this monkey was less susceptible to the syphilitic virus than the long-tail varieties, might fill the necessary requirements. Two rhesus monkeys, which were the subjects of experimental syphilis, were sent from Finger and Landsteiner, of Vienna. One of these was the eighth, the other the ninth monkey of a series which had been successively inoculated. The monkey of the eighth passage presented well-marked lesions; that of the ninth passage showed only the slight remains of a primary lesion. A chimpanzee inoculated with material from the two rhesus monkeys developed well-marked primary and secondary lesions, so that it was evident that eight passages through the rhesus monkey is not sufficient to attentuate the virus for higher apes. The virus was then inoculated from the chimpanzee through twenty-two rhesus monkeys. During these inoculations the lesions in the monkeys were observed to change in certain particulars. The primary lesion was found much earlier, appearing in the last few monkeys on the seventh instead of the nineteenth day, while the infection was of a much less severe nature. But the most important result was the attenuation of this virus for other monkeys. With inoculation of this virus the Javanese macacus developed very slight primary lesions after twelve days' incubation, while the chimpanzee, the most susceptible of all monkeys, proved entirely refractory.

They further relate certain observations made upon one of their assistant janitors, who developed a small round ulcer upon his lower lip. Most of the janitor's time was employed in caring for and handling the inoculated monkeys. Following the sore there was no glandular enlargement. The man feared that he had acquired syphilis, and though the lesion did not resemble exactly a chancre he induced Metchnikoff and Roux to make a complete investigation. To their surprise, in scrapings from the sore, they found typical spirochetæ of Schaudinn, and monkeys inoculated with material from the sore developed unmistakable syphilis. The man then consulted Fournier, who could find nothing to justify the diagnosis of syphilis. For six months the man was carefully watched, but during this time did not develop secondary symptoms. The virus from this man was then inoculated into twenty animals, three chimpanzees and seventeen monkeys, representing in all five species. Of these, the three chimpanzees did not develop secondary symptoms. This suggested that the virus from this particular man was of an attenuated variety. When this virus, however, had once produced syphilis in monkeys it was capable of protecting the animals against a virus from other human sources. Metchnikoff and Roux believe that the man acquired his lesion from inoculated monkeys, and that the atypical character of the sore and mildness of the infection were due to the fact that the virus had been attenuated by passages through the monkeys. It could not be determined whether or not the man had been vaccinated against human virus, for he refused to be inoculated with material from human sources. The effects produced by the inoculation of virus from monkeys was, however, studied on one man who consented to undergo the operation. The virus had been passed through five Simian monkeys. Inoculations were made upon the arm of the man and at the same time upon the eyebrows of a chimpanzee. The chimpanzee developed a typical, primary lesion followed by swelling of the lymph glands, while the man, after an incubation period of twelve days, showed, at the site of inoculation, a crop of small papules which did not ulcerate, and which disappeared after a few days. In view of the great difficulty in determining what late effects may follow vaccination of human beings with virus attenuated by passages through monkeys, Metchnikoff and Roux do not believe much can be hoped for at present from this method of possible prevention. Until further experiments can be made they emphasize the importance and value of the use of calomel ointments as a prophylactic measure and cite cases to show that the contraction of the disease, which was almost certain to have occurred, was prevented by this method of treatment.

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THE

AMERICAN JOURNAL

OF THE MEDICAL SCIENCES.

APRIL, 1907.

ORIGINAL ARTICLES.

THE MEDICAL STAFF AND ITS FUNCTIONS: A STUDY IN HOSPITAL ORGANIZATION.

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Introduction.—The problem of medical organization is one of adjustment of means to ends. In order to deal intelligently with the subject of medical organization we must begin with a clear understanding of the purposes of the hospital which the medical staff serves. A preliminary consideration of hospital function will point the way to the proper form of medical organization, and will save us from the pessimistic bias of Dr. Worcester, who regards the "medical question" as the "bugbear of hospital management."[1] What, then, are the functions of the hospital?

First, we have to regard the hospital as the servant of its patients—as an institution which exists primarily for the cure of disease, for the relief of suffering, and for the promotion of the physical welfare of its patients. The medical organization of the hospital must, therefore, be based mainly upon a consideration of the needs of its patients, whether as applicants for admission, as inmates of the wards, or as convalescents.

Second, it is apparent that each hospital, by its methods and practice, is able either to further or to retard medical education and the march of the medical sciences; and since the comfort and prospects of hospital patients depend upon the development and dissemination of medical knowledge, we cannot dismiss the subject of the medical organization of hospitals without endeavoring to ascertain its proper relation to progressive medical education.

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[1] A. Worcester, A.M., M.D., Small Hospitals, p. 29.

Third, it is apparent that the work of the hospital is limited by its resources; and we are thus led to weigh carefully the economic bearings of our subject. Having in mind the primary purpose of the hospital, we shall be prepared to sanction all those expenditures connected with or influenced by medical organization, which yield a return of human life and comfort. We wish, however, to know what these expenditures are, and how they affect the results aimed at; and we desire so to regulate them as to avoid all unnecessary waste.

Fourth, we must deal justly with the men who give to the hospital the thought and effort of their best years: this for the sake of the hospital, as well as in obedience to the moral law; for if the avenues of professional growth are cut off from the medical staff, if their lives are shortened or their success hampered by worry or overstrain, the cause of the hospital will suffer.

We have, then, four general subjects for consideration: (1) The medical staff in relation to the needs of the patient; (2) the medical staff in relation to medical education; (3) the medical staff in relation to hospital economy; and (4) the medical staff in relation to the rights of its members.

I. THE MEDICAL STAFF IN RELATION TO THE NEEDS OF THE PATIENT.

The Consideration of Applications for Admission. One of the first necessities in medical organization is a system of examining candidates for admission to the wards of the hospital which will guarantee the fair and intelligent treatment of all who apply, and which will provide for the immediate and effective handling of all emergencies which require attention on the spot. The demands of patients who seek admission to the hospital are many and varied. The admitting officer will find himself called upon to decide upon the relative claims of a large number of candidates competing for admission to a limited number of beds; to exclude contagious cases in which the condition of the patient justifies transfer; to give sound advice to rejected applicants; to maintain, in the interest of rejected applicants, amicable relations with various relief agencies; and to develop a habit of reciprocity with the officers of neighboring hospitals, homes, and asylums.

The admitting officer must be guided in his work by the accepted policy of the hospital. But while he is compelled to recognize that in the admission of patients the hospital is subject to certain restrictions of space and of classification, he will nevertheless remember that illness is often so sudden and uncompromising in its attacks, and that the machinery of relief at the disposal of the hospital is so elastic, that blind or rigid adherence to any set rule governing the admission of patients may work needless hardship and may even result in avoidable loss of life.

It is a function of the medical organization of the hospital to provide an admitting officer whose discretion and capacity will give effect to the spirit and purpose of the law, rather than to its naked letter; only through a physician of first-rate ability and character can the complex and often vital interests of applicants for admission be safeguarded. If the duty of admitting patients to the hospital is assigned to relatively inexperienced internes, who at the same time are burdened with heavy responsibilities in the wards, the wise selection of cases for admission cannot be ensured and the problems arising out of the rejection of applicants cannot be satisfactorily solved. Here, if anywhere, rapid changes in office, especially such changes as imply the frequent introduction into the admitting department of youthful men of immature judgment, are to be shunned. In the case of a large hospital the admitting physician must be ready to put aside the claims of private practice and to devote a large part, if not all, of his time and energy to the hospital; and the trustees should not hesitate to pay him a salary commensurate with the services demanded.

To state the matter briefly, we may say that, as a rule, the office of admitting physician should be filled by a salaried man, professionally mature; in the case of a large institution a resident admitting physician is desirable; internes holding office for a short time only are not equal to the delicate tasks which the admitting physician is called upon to perform, and are best excluded from the admitting department altogether, unless the hospital is provided with a medical superintendent competent to supervise closely the work of the house staff.[2]

THE GROUPING OF PATIENTS AND THE DANGERS OF SPECIALISM. While the classification of patients and their assignment to the various departments of the hospital is the duty of the admitting physician, he must take the wards and departments of the hospital as he finds them. Now, in many instances, the treatment of a hospital patient is governed, perhaps more than is wholly desirable, by the special aims and practice of the department in which he happens to be placed. In the official grouping of patients and the corresponding assignment of attending physicians and surgeons, therefore, the hospital should seek to avoid any undue exaltation of specialism; and yet there must be at least so much emphasis put upon the exploitation of special fields of medical and surgical endeavor as will encourage the development of the finest skill in diagnosis and ensure the employment of such particularized methods of treatment as have been shown to yield the best results.

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[2] In view of the fact that many hospitals today are content with the examination of applicants for admission by the younger members of the house staff, it is interesting to read, in Dr. Morton's History of the Pennsylvania Hospital, that when in 1802 the physicians of that institution pleaded to be allowed to transfer to their pupils the duty of examining applicants for admission, on the ground that numerous engagements frequently made it impracticable for the physicians to do the work themselves, their petition was promptly and firmly denied. The reply of the Board was as follows: "The Managers are all of the opinion that they cannot dispense with any part of the present form of admission. The Board regret that the physicians have made a request which the Managers cannot grant without infringing on the duties they owe the institution."

A hospital the clinical departments of which are too minutely subdivided and classified, and of which each subdivision is a segregated and self-governing unit, ceases to be a satisfactory or even a safe instrument for the relief of its patients. For example, to assign a woman to a gynecological ward, where in the hurry of specialized routine she will either be made the subject of an immediate operation, or without further consideration will be dismissed from the hospital as an unsuitable case, is not the best treatment for all patients who at first glance appear to be proper subjects for operative gynecological treatment. In many such cases, further study would reveal a complex of pathological conditions, some of them susceptible to and clearly indicating medical treatment of a kind which the average busy and successful regional surgeon, whose horizon has inevitably contracted in consequence of years of concentrated devotion to a restricted field, does not habitually practise. The psychological tendencies of specialism, therefore, furnish one reason for establishing such sweeping oversight of the various clinical departments as will promote their active, liberal, and persistent coöperation. If possible, we must find the means by which such general control can be provided.

THE FUNCTION OF THE CHIEF PHYSICIAN AND CHIEF SURGEON. A single chief in command of the medical division, and an officer of similar rank in command of the surgical division, each exercising general supervision over all the wards of his department (however these wards may be subdivided or specialized), can do much to enhance the efficiency of the visiting staff and its subordinates. Under such control the patient is regarded as a ward of the hospital, having a broader claim than the patient whose professional guardian is an individual attending physician or surgeon. The grouping of many wards under one recognized head produces a community of clinical interest which is far better for many patients than the rigid maintenance of finely drawn department lines. Free trade within the surgical or the medical division, and the liveliest reciprocity between the medical and the surgical departments, are essential to the welfare of the patient. By the very character of the medical organization, patients should be guaranteed thorough treatment as human beings, and not left to the restricted interests of individual practitioners.

TREATMENT OF PATIENTS IN THE WARDS: NUMBER AND DUTIES OF VISITING PHYSICIANS. Assuming that the patient has passed the admitting office, and has become a member of a divisional or ward group, let us inquire into his further needs, and the means furnished by the medical organization for meeting them. On his admission to the ward, the patient's first claim is for prompt attention from the physician who is to be mainly responsible for his care. The preliminary inspection of the patient should be made at once by the senior house officer; and as soon as the history of the case has been recorded and the physical examination and routine scientific investigations made (or even before these if the case is urgent), the attending physician or surgeon should promptly appear on the scene.

A proper organization will provide a sufficient number of competent attending practitioners to ensure prompt and efficient attention to every patient. Let the chief of division control as many wards as you please—the more the better; for the attending physician or surgeon, however, there should be only as many beds as he is able to keep under his eye during every day of his official duty. Once in every twenty-four hours at least the visiting officer should see every patient, and as much oftener as the circumstances of the case demand. It is dangerous to trifle with this daily rule, especially in hospitals which deal largely with acute cases. To leave the matter entirely to the judgment of the physician, or to require him to see acute cases no oftener than two or three times a week, is to sanction the negligence of the physician whose conscience gives way when the claims of private practice press hard.

Compulsory daily visits, then, should be the rule; and if this seems to bear too heavily upon men who give their services to the hospital gratis, and who are obliged to earn their bread outside of the institution, let us lighten their burden not by the omission of the attention to which the patient is entitled, but by appointing an adjunct or associate to take the place of each regular member of the visiting staff when necessity arises. The daily attendance book, in which is recorded the day and hour of each visit of every member of the visiting staff, is a true index of the hospital's vitality, and of the devotion to duty of its physicians and surgeons.[3]

COMPOSITION, DUTIES, AND TRAINING OF THE HOUSE STAFF. While a daily visit on the part of the attending physician or surgeon suffices in ordinary cases, two visits daily to the bedside of every patient constitute an irreducible minimum for the senior interne of the division. This need must be kept in mind in deciding upon the number of patients to be included in one department; and the requisite number of visits should be guaranteed by a rigid regulation, to which no exception should be tolerated.

If in addition to the senior resident officer of the division and his first assistant or prospective successor, the house staff includes at least one physician, interne or externe, for every ward unit of from twenty to thirty beds, the patient will be in a fair way to receive proper routine medical attention.

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[3] The Committee on Reorganization of the Medical Staff of the Toronto General Hospital reports Dr. Osler as recommending that "the physicians should not only make their visits at stated hours but should *stay* stated hours."—National Hospital Record, June, 1906, p. 21.

In many representative hospitals, the duration of service of internes has been fixed at two years. It is a wise rule which divides equally between the medical and surgical wards the work of all new men during their first year in the institution, whether they are destined for ultimate promotion in the surgical or the medical service. Breadth of clinical experience is a desideratum of the house officer, as well as of the visiting physician and surgeon. The house physician who has served in the operating room knows the surgical point of view, which is different from his own; and he not only appreciates the utility of prompt surgical treatment in suitable cases, but estimates sanely the limits of surgical aid. Lacking actual experience in the operating room, his understanding would never be equally enlightened.[4]

The presence of the entire staff of the division should be exacted at general rounds each day. This will tend to lessen the number of examinations to which the patient is subjected, while giving each member of the staff opportunity to follow every phase of each case in his division. At the same time it will encourage an interchange of ideas which may result in benefit to the patient.

RESPONSIBILITY OF THE SENIOR HOUSE OFFICERS NOT TO BE SHIFTED. The necessity for strict control of the work of the house staff will appeal to every experienced hospital manager. No physician should be made responsible for a patient, whether in the wards or on the ambulance, during the first year of his hospital service; and the absence of senior residents, even for a short period, should not be tolerated if it necessitates a transfer of responsibility to the shoulders of inexperienced men.

NO PROMOTION WITHOUT PROVED FITNESS. As the house officer advances, step by step, from the lowest to the highest position on the temporary resident staff, it may reasonably be demanded of him that he prove his fitness for promotion. However honestly, faithfully, and intelligently the examination may be conducted which gives the house officer his first foothold in the hospital, unfit or undesirable men from time to time will secure places on the staff. If it is understood at the time of appointment that each sub-period of service will be regarded as a period of probation, and that there will be no promotion without previous proof of fitness, better discipline and more sustained effort will result. Time and again the life of a patient rests in the hollow of the hand of the house physician or house surgeon. The hospital should see to it, therefore, that its responsible house officers are fitted by character and training to act in all emergencies.[5]

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[4] The following rule prevails in Mt. Sinai Hospital: "Appointments to the house staff are made for a total period of two and one-half years. Appointees are assigned for service to either a medical or a surgical division; but surgical internes are required to spend six months in the medical wards, while medical internes are assigned for six months to surgical wards."

THE NEED AND EFFECT OF DISCIPLINE. In the assignment of duties, the exact responsibility of each member of the staff should be so defined that blame for neglect or inefficiency will automatically be put where it belongs and credit given where credit is due. It is, of course, conceded that in an organization where the results achieved are often due to concerted group action rather than to the efforts of an individual, exact justice cannot always be done in the allotment of credit to each member of the group; and yet it is of the utmost importance to lay bare, as far as possible, the weakness of those who contribute little or nothing, and to reward, at least with moral support, the conscientious and useful worker. By following this course the hospital aids in the intellectual and moral growth of its house staff.

FACILITIES FOR SCIENTIFIC INVESTIGATION. In an organization as highly differentiated as that of the modern general hospital, there is necessarily much interdependence among the groups of workers. The efficiency of house staff and visiting staff alike is largely dependent upon the facilities afforded for scientific investigation. In the patient's interest, therefore, there must be provided a competent pathologist, exercising supervision over the pathological and bacteriological laboratories, and having some measure of control over all the laboratory work of the house staff.

Without considering, in detail, the organization of the pathological department, let me say that the usefulness of the department can be enhanced by throwing open its doors under suitable conditions to volunteer workers. In the larger cities volunteers are always available; their labor can be utilized at small cost to increase the scientific output of the hospital and to contribute to the thoroughness of the work done for its patients. The most desirable recruits for this work are men who have served on the house staff of the institution and who desire to supplement their clinical training by a period of concentrated laboratory work.

The visiting staff should have the privilege of assigning to the pathologist or his assistants all important laboratory work required in the interest of hospital patients; but in some instances visiting physicians will prefer to undertake laboratory investigations themselves, and the director of laboratories should be required in such cases to offer every possible opportunity for the proposed work.

SPECIALISTS AS CONSULTANTS. Reference has already been made to the position which should be occupied in a general hospital by the so-called medical and surgical specialties. While there is danger in the multiplication of independent departments for such work, those branches which have been highly elaborated by the endeavors of special workers and the recognition of which is approved by current practice, should have due consideration. If distinct clinical departments are organized for the practice of these branches, they should be co-ordinated with the medical and surgical services, by placing them under the direct control of divisional chiefs. If specialists are not included in the regular visiting staff, the way should be made easy for the utilization of their talent and skill by naming them as a consulting or advisory staff. When engaged in the work of the hospital, consulting specialists should rank with the regular visiting physicians, and should be subject to similar control by a divisional chief.

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[5] From the rules of the Boston City Hospital I quote the following: "It is, however, in all cases provided that after one term of service the promotion of a House Officer for the succeeding term of service shall be dependent upon the satisfactory performance of his duties in the department to which he has been previously assigned, and failure to satisfactorily perform his duties shall subject such House Officer to removal from his position."

Recognition of the Family Physician. There is another form of consultation of which it is pertinent to speak here—a form which is not generally favored by hospital men, but which from the standpoint of the patient is often desirable. I refer to consultation with the patient's own physician, who has previously studied the case outside of the hospital. The family physicians of hospital patients should be more frequently seen in the wards, not for the purpose of enlightening the hospital staff, but for the satisfaction of the patient himself.

The family physician who is not officially attached to the hospital should be given free access to the institution whenever his professional zeal or his sympathy for his patient prompts him to come; and he should be not only tolerated, but warmly welcomed when the patient expresses a desire to see him. It is impossible to insist too strongly upon the duty of the hospital to recognize, at least in this unofficial way, the right of the patient to the counsel and cheer of his familiar adviser, and also the right of the family physician to follow his patient after the latter has been admitted to the hospital.

The Period of Convalescence. We have thus far traced in outline the needs of applicants for admission to the hospital and those of inmates of the wards, and have considered briefly the kind of medical organization which these needs indicate. We arrive now at the stage of the patient's discharge.

It is the duty of the attending physician to see every acutely sick patient in his wards during each day of his service. This duty, however, does not cease when the patient enters upon the stage of convalescence, but continues to apply up to the very moment of his discharge. Indeed, the attending physician who does his full duty will acquaint himself with the plans for the immediate future of the patient who is about to be dismissed, and will endeavor by timely advice to prevent avoidable errors in conduct. Ignorance of their bodily needs is no less characteristic of hospital convalescents than of the poorer classes generally, and to prevent disease is no less the function of the hospital physician than to cure it.

Care after Discharge: Unity of Hospital and Dispensary. The day of the patient's discharge from the hospital is a critical one. If continued treatment is necessary, it is the duty of the attending physician to see that the patient, if able to pay, is safely guided from the hospital into the care of his family physician, or, if a suitable subject for dispensary treatment, is directed to the proper division of the outdoor department. By a proper subordination of the physicians of the dispensary either to the attending staff or to the divisional chiefs of the hospital, the interchange of clinical records will be facilitated, thus enabling the patient to have the benefit of continued treatment under a single medical authority. The slipshod character of the work done in dispensaries attached to hospitals famous for thorough indoor work is often due to the separate medical organization of the dispensary, and to the fact that the service in the latter receives no suitable recognition at the hands of the hospital authorities. Shorn of hospital privileges, the dispensary staff is prone to lose professional caste; the best men do not continue in its service, and the quality of its work deteriorates.

I repeat, therefore, that unity of control of indoor and outdoor services is essential to good hospital government. The method of binding the two departments may vary in its details, but a form worthy of special consideration is that adopted by hospitals whose surgeons to out-patients serve alternately as assistants to the visiting surgeons of the hospital. Under this system the visiting surgeons have a double staff of assistants, one staff being occupied in the wards while the other is on duty at the dispensary.[6]

Executive Control of the Discharge of Patients. If the interests of the patient require to be safeguarded in the admitting-room and in the wards, it is no less necessary to impose a reasonable check upon the discharge of patients from the hospital. Neglect of the rights of patients awaiting discharge is not creditable to any hospital, for mistakes made at the last moment may undo the good of weeks of careful treatment. In large hospitals the supervision of recommendations for the discharge of patients may profitably be entrusted to an executive officer, preferably a medical man, whose business it should be to keep in close touch with all the patients in the hospital. He should be familiar with all the charitable relief agencies in the community which are capable of dealing with any phase of the physical or social needs of former hospital inmates, social needs being here included because of the frequent interdependence of social condition and physical ailment.

The Patient's Interest in Medical Education. In considering the medical organization of the hospital we have thus far kept in view the immediate needs of the patient. Before passing to the question of the relation of the staff to medical education, we should perceive that the new point of view is not wholly dissociated from that of the patient's interest. Hospitals which are engaged in the promotion of medical education are bound to maintain high standards of professional achievement; their staffs enjoy the inspiration of teaching and are compelled to keep abreast of the times. Such hospitals are constantly in the public eye, are subject to the keenest criticism from without and within, and, owing to the rivalry among teaching institutions, are bound to favor any policy which will enhance their efficiency. So manifest are the benefits derived by the inmates of a hospital from the school atmosphere, that hospitals which endeavor to promote medical education need not justify their policy, however readily they might do so, by pointing to its good effects on later generations and in distant communities—effects which will be dwelt upon in the following section.

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[6] For details of a method of uniting the clinical practice of hospital and dispensary, consult the rules of the Massachusetts General Hospital.

II. THE MEDICAL STAFF IN RELATION TO MEDICAL EDUCATION.

The Misson of the Hospital. If it is true, as Dr. Withington[7] has said, that "the mission of the hospital is to the sick poor of every country and of all time," it will be well to bear in mind that sufferers outside of the hospitals are benefited by the training of physicians, by the advancement of pathological knowledge, and by the elaboration of therapeutic methods in hospital wards and laboratories. Accepting Dr. Withington's interpretation of the mission of the hospital, we are bound to recognize the training of physicians, the promotion of scientific medicine, and the dissemination of its teachings, as legitimate hospital functions.

What is a Teaching Hospital? Hospitals are frequently spoken of as "teaching hospitals" or "non-teaching hospitals." The principal teaching hospitals are those associated with medical schools; but any hospital which admits undergraduate students to its wards and laboratories, and even those which provide an instructive course of work for medical graduates, may claim to rank in the same class. In many hospitals, recent years have witnessed a striking growth in the house staff, and the number of highly trained practitioners has thus been largely increased. Only sixty years ago in the Pennsylvania Hospital there were but 2 resident physicians who served two years. Each of these took charge alternately of about 200 insane patients, male and female medical wards, obstetrical wards, and male and female surgical wards where there were frequently 80 patients to be dressed each day. Today in the Pine Street branch of the Pennsylvania Hospital alone there are 9 resident physicians, while the department for the insane numbers

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[7] The Relation of Hospitals to Medical Education.

5 more. Mount Sinai Hospital, caring for about 400 patients, is provided with a house staff consisting of 32 recent graduates in medicine; furthermore its pathological, bacteriological, and chemical laboratories furnish work for 6 volunteers, in addition to 7 regular appointees of the department; its outdoor department employs 106 physicians, half of whom are in attendance each day. And these large figures do *not* include the regular attending and consulting staff.

It is not necessary to point out the many ways in which the welfare of patients has been promoted by the growth of the house staff, which has been one of the characteristic developments of the past generation.

THE PRIVILEGES OF STUDENTS IN HOSPITAL WARDS AND THE NATURE OF CLINICAL EXPERIMENTS. It is the boast of some hospitals that students are rigidly excluded from their wards; and this policy is intended to appeal to the prejudice of hospital supporters who regard the presence of students as inimical to the welfare of patients. But is not such prejudice due to mistaken ideas concerning the functions and privileges of medical students in hospital wards? Inquiry will show that a widespread belief exists that students are permitted to do very much as they please with hospital patients to whom they have access; that dangerous experiments are tolerated; and that needless suffering, if nothing worse, results.

Of course, no one in his right mind would willingly subject himself to reckless medical experiments. On the other hand, the physician who in his private or hospital practice never makes an experiment based upon reasonable deduction from known facts, or upon results empirically obtained by other competent professional observers, does not fully grasp his opportunities or responsibilities.

Rational and legitimate experiments in therapy, and constant study of the sciences underlying the treatment of disease, mark the progress of every practitioner who is successful in the better sense of the term; if the presence of students as witnesses of such experiments alters the situation at all, it does so in a manner which is beneficial and not injurious to the patient. The presence of students admonishes the physician to exercise his best care and judgment, since to fail before this critical audience is to suffer injury to his dearest possession, that is, his professional reputation.

If it is once made clear that the presence of students does not necessarily result in subjecting the patient to needless and cruel experiments, there remains only the familiar objection to overfrequent examination, a vice which is supposed to be peculiar to the college hospital. But by the very refusal of a majority of the hospitals to open their doors to students of medicine, encouragement is given to the introduction of unwieldy crowds of learners into the limited wards of the few hospitals which maintain an educational policy. Once bring all of the hospitals of the community into proper relation with the medical schools, and the necessity for overcrowded clinics disappears.

There is, after all, no great difference between admitting advanced undergraduate students to the wards, which few hospitals are willing to do, and accepting as members of the house staff men who enter as graduates after completing four years of theoretical study, which is the common policy. In greater and greater numbers recent graduates are obtaining recognition as part of the hospital staff; this phase of hospital development will only be completed when the law requires every man who desires to enter upon the practice of medicine to show credit for at least one year of hospital work, and when hospitals so modify their organization as to provide places for a reasonable number of non-resident undergraduate pupils who will carry into private practice the results of an experience for which no substitute exists, and whose presence in the hospital will stimulate the attending staff to greater effort and will thus increase immeasurably the power of the hospital for good.

Control of House Staff by Medical Superintendent. As the house staff grows from year to year, and as undergraduate students take their place beside the members of the graduate staff, the need of strong government increases. For administrative purposes it is wise to subordinate the house staff directly to the superintendent of the hospital, if the latter is a medical man, and to emphasize his authority by the title of "Resident Physician and Superintendent" or "Medical Superintendent." The value of such control increases in proportion to the size and complexity of the hospital. Especially in hospitals where teaching is systematically done, an administrator of professional training, familiar with the aims and methods of medical education, in sympathy with its ideals, and yet cognizant of the inviolable rights of patients and awake to every avenue through which abuse may enter, will prove of inestimable value to the college faculty and a bulwark of safety for the patients.

The Training of Nurses. The admission of medical students to the wards of the hospital or, as a substitute for this, the organization of the resident staff upon a liberal and systematic scheme, does not complete the educational work of the hospital. While the training of nurses is of relatively minor importance, this training can be carried out nowhere else so effectively or economically as in a hospital. If hospitals have been readier to accept an educational function with reference to nurses than in connection with the teaching of medical men, it is because trustees early perceived the economical advantage to the hospital which lay in its association with the training school for nurses.

Lack of time forbids extended discussion of the methods of the hospital training school. In passing, I shall only express the opinion that, having undertaken this educational work, hospital managers owe it to their pupils to provide an efficient teaching staff, whether paid or unpaid. Furthermore, the time and strength of the nurse must not be exhausted in the performance of menial or routine duties which form a proper part of her work, but which need to be supplemented by theoretical instruction, if the nurse is to enter properly equipped on the private practice of her profession.

The Place of Medical Records in the Promotion of Medical Science: The Registrar. We must not overlook the value to medical education and to the promotion of medical science which lies in the careful preparation of clinical records and in the intelligent compilation of statistical reports. Some attempt should be made to discover, through the study of hospital records, the causes of disease and the means of its prevention. To leave the valuable records of the hospital to the chance interest of untrained juniors of the house staff or to the leisure and whim of uncomprehending clerks is a fatal error. That hospital records and clinical statistics are used so little, and that the best of them are of so little use, is attributable to the rarity of competent hospital registrars. Widespread recognition of the office of medical registrar in the scheme of hospital organization would produce an improvement in the treatment of hospital statistics from which contributions to preventive medicine and to the knowledge of disease in its social aspects would inevitably be derived.

The Grouping of Patients and its Influence upon Systematic Study. Touching the relation of the visiting staff and its organization to medical education, let me say, at the risk of seeming repetition, that if the fragmentation of the hospital service is avoided and the medical and surgical divisions respectively are grouped under single heads, the systematic study of cases and rational experimentation will be facilitated. The number of patients to be assigned to each attending officer should be so limited that a thorough study of every case may be reasonably assured.

Relations between Visiting Staff and Pathologist. In order to guarantee proper coöperation between the visiting and the pathological staff, the pathological department should be under the control of the divisional chiefs. The chief pathologist, whether resident or visiting, should receive a fair salary, because he cannot derive from his work in the hospital the pecuniary advantages which accrue from their hospital connection to attending physicians and surgeons. In return for his salary, the pathologist should be required to give to the work of the hospital as much time and personal effort as may be required to work up properly the material assigned to his department. In the performance of this duty, he is entitled to the aid of a reasonable number of assistants.

Connection of Staff with Other Hospitals. I believe that, contrary to the rule of some hospitals, the association of attending physicians and surgeons with medical schools and with other hospitals should be encouraged for the purpose of multiplying the sources from which knowledge applicable to the work of the hospital may be derived; but no such association with its consequent demand of time and service should be accepted as an excuse for the neglect of hospital duty.

Objection to Rotation in Ward Service. In order to extend the privileges of the hospital to a wide circle of the medical profession, it is advisable to limit the number of beds assigned to individual attending physician and surgeon and provide a substitute or adjunct for each. The superiority of this plan over one which includes larger individual services with frequent rotation of duty is due to the fact that frequent changes interfere with that continuity of study and observation which best promotes discovery.[8]

If the foregoing recommendations are adopted, the hospital will be in a fair way to enlist in its work the students, the thinkers, the leaders of the medical profession. Having secured the co-operation of these powerful factors in its work, it should treat them in no niggardly manner, but with a cheerful spirit should place in their hands the means to carry forward their life work.

III. THE MEDICAL STAFF IN RELATION TO HOSPITAL ECONOMY.

From the standpoint of hospital economy the organization of the medical staff may be studied with regard to its effects upon hospital income and upon hospital expenditure. The most casual observation reveals the many ways in which the daily labors of the medical staff are bound up with hospital expenditure. The connection between medical organization and hospital income is less intimate and less apparent, and yet it is by no means negligible; for example, hospital appointments and privileges constitute not only a possible, but in some instances an actual, source of income. Further sources of income which require regulation and development are the collection of fees from medical students and medical colleges, and charges made to private patients.

The Sale of Staff Appointments as a Source of Income. Unfortunately, the sale of hospital appointments is not unknown. It is more especially the expedient of small institutions, whose ambitions soar above their means and who have yet to learn the superiority of patient and painstaking clinical achievement over momentary spectacular financial success. The practice of selecting candidates for hospital appointment chiefly with a view to increasing the income of the hospital by levying upon the fortunes or the influence of appointees is fraught with danger, because it opens the door to wealth while it bars out worth and talent. The true success of a hospital is measured by the good accomplished and not by the assets accumulated, and the only admissible test of fitness for appointment is ability to do the work and to promote the interests of the institution. Even if commercial standards were admissible, I believe it could be shown that in the long run the services of a visiting physician of marked ability will do more to enhance the popularity and increase the wealth of an institution than any contribution of ready money linked to the work of an unfit man.

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[8] Dr. A. G. Gerster, The System of American Hospital Economy, Medical Record, December 2, 1905, advocates continuous service as against rotation in service, on the score of *economy* as well as of efficiency.

THE PAYMENT OF FEES BY MEDICAL STUDENTS. In one form only is the acceptance of money from medical workers justifiable, and that is when fees are paid by medical students in whose interest facilities for teaching are maintained. As long as the number of hospitals to which students are admitted is limited, those which open their doors to undergraduates will find their participation in medical education costly. As an illustration of this let me call attention to the problem facing the managers of a hospital which is soon to be built. The trustees of this institution have been informed by their medical adviser that if a teaching hospital is built—that is, one having amphitheatres, demonstration-rooms, lecture-rooms, cloak-rooms, and students' laboratories—the available fund will produce a hospital of only two hundred beds; whereas the elimination of teaching facilities would enable the committee to erect a hospital of three hundred beds.[9]

The large relative cost of a teaching hospital today is due to the fact that teaching hospitals are so few, necessitating extraordinary expenditures on the part of the handful which undertake a duty that really belongs alike to all. But taking the situation as we find it, is not a hospital justified in stipulating for the replenishment of its treasury by those who, not being objects of charity, help to deplete its funds? If so, the hospital is entitled to a fee from every undergraduate student who enters its doors.

The thought suggests itself, that if the privileges afforded to the non-resident students are valuable, of how much greater value are the far-reaching privileges which are open to the resident house staff, who come to the hospital fresh from college, bursting with book knowledge, but wholly lacking in clinical experience? These men, however, give all their time to the hospital, and the service exacted of them offsets their privileges. The exaction of fees from the regular house staff would, therefore, be inequitable, though I do not doubt for a moment that in declining to enrich itself at the expense of its staff the hospital renounces a handsome income.

PRIVATE PATIENTS AS A SOURCE OF INCOME. We have next to consider the bearing of medical organization upon the admission of private patients, from whom many hospitals derive a large part of their income. In hospitals which combine private and public wards, the organization and distribution of the staff, the number of appointments made, and the extent to which hospital privileges are accorded to irregular members or non-members of the staff, have an important effect on income. In the adjustment of these matters, at least in hospitals which exist primarily for charitable work, the chief consideration must be the welfare of the patients in the charity wards.

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[9] The Brigham Foundation, Boston, Mass.

I have no doubt that any superintendent who chose to do so could largely augment the income of his institution by encouraging applications from patients able to pay and discouraging the admission of patients unable to do so. This could be accomplished with particular ease if the hospital followed the policy of one of the oldest hospitals in the East, the members of whose staff are compelled to give their services gratuitously to all the patients of the hospital, whether occupying charity beds or high-priced private rooms. The result of generally introducing this policy would be to ruin private hospitals and sanitariums; to curtail the legitimate incomes of medical men; to crowd out of mixed hospitals a large number of poor patients unable to pay either for hospital accommodations or for treatment; to give free treatment to persons who do not deserve it and in many instances do not even desire it; and, finally, we must admit, to add large sums to the incomes of semi-private hospitals. But, happily, the encouragement of the *business* of the hospital, that is to say, the careful fostering of its pay patients' account at the expense of its charity work, is not the sort of social service in which most of us are engaged. Our hospitals are primarily charitable institutions, and we must not, in their management, acknowledge subserviency to business principles which do not properly apply to the work in hand.

Appointments to the Regular Staff Not to be Governed by the Needs of Private Wards. The organization of the medical staff of the hospital proper, by which I mean the public wards, must not be sacrificed to the business needs of an overgrown department for private patients. The test of the worth of a hospital physician is his ability and faithfulness in caring for the hospital's charity patients; if in addition to this he is able to send many patients to the private wards, well and good; if not, his position on the staff must not be jeopardized. While, as a rule, private wards will not yield any considerable surplus revenue, yet they ought financially to take care of themselves; and in planning the medical organization of this department the hospital will do well to extend its privileges to a circle sufficiently wide to guarantee a self-supporting department. This can be done and should be done without prejudice to the charity service.

From time to time hospitals will find themselves in the dilemma of a hospital well known which, having erected a large building for pay patients, found that the maintenance of this building was so costly as to constitute a drain on the treasury. Private wards half empty did not pay; and for its own protection the hospital was compelled to abandon the restrictions formerly placed upon the treatment of patients in its private wards. These privileges, originally confined to the regular attending physicians and surgeons of the charity wards, were now extended to former members of the house staff and to older members of the dispensary staff, and the financial strain was thus relieved. Observe, however, that in this instance the difficulty growing out of an overbalanced private department was overcome without in any way interfering with the medical organization of the hospital proper, the characteristics of which must be determined by the needs of the chief class of its patients, and without regard to financial considerations.

THE RELATION OF THE VISITING STAFF TO HOSPITAL EXPENDITURE. Having thus briefly alluded to the points of contact between medical organization and hospital income, we may pass on to the subject of hospital expenditure.

It is customary to assume that the attending staff is careless in regard to the use of hospital property, that its example is communicated to the house staff and nurses, and that hospital expenditure is thereby increased. While there is some truth in these suppositions, the underlying cause is disregard of the principles of economy in the medical organization of the hospital. Is it not a blunder to cut off, by an artificial gulf, the professional conduct of the hospital from its business administration? The functions of the two departments are not only complementary—they shade into one another and are not practically separable.

Much can be gained for economy in the use of supplies by providing a place in the medical hierarchy for the chief executive officer of the hospital—an arrangement which, as already pointed out, is desirable on other grounds also. But this inclusion of the chief resident administrative officer in the medical system of the hospital is only a beginning; it places him in close official contact with the medical staff, enabling him discreetly and without offence to admonish them of their oversights.

THE PRESENTATION OF ECONOMIC REPORTS TO THE MEDICAL BOARD. Of greater importance is a clear understanding and a systematic use of the economic facts in regard to medical administration. It is not reasonable to expect visiting physicians and surgeons to devote their time to a study of the details of hospital expenditure, a study foreign to their daily thought; but it is both reasonable and just to require them to give at least so much time to the subject as may be necessary to familiarize themselves with facts ascertained through systematic records, showing in detail, and by comparison of ward with ward and department with department, the amount expended for all supplies which are used in the discretion of the medical staff. These facts, thoroughly digested and graphically described, should be presented at regular meetings of the medical board of the hospital, accompanied by pertinent recommendations by the lay board or superintendent. Why may not the reading of such reports constitute a routine part of the monthly business meeting of the medical board, so that there can be no shirking of the duty of preparing the reports and no chance of overlooking their significance? This is but one phase of that coöperation with the chief executive officer of the hospital which should be kept constantly in mind and which should be encouraged to the utmost in the determination of official relations.

The Item of Medical Salaries. With every medical appointment there should go a suitable reward for the service exacted. It is a mistake to lean too heavily upon the supposed altruism of the medical staff. The beginner who enters the service of the hospital fired with zeal for mankind may find his ardor cooled by the contemplation of the material needs of a growing family. While participating willingly in the work of the hospital, he cannot shirk his responsibilities as a citizen, husband, or father, nor is it right for the hospital to belittle these claims.

For the attending physician, who gives only part of his time to the work of the hospital, and who secures in return opportunities for study and teaching and prestige associated with these, there need be no question of salary; for the house staff, too, the professional rewards are surely commensurate with the sacrifices made; but medical men who devote all or a large part of their time either to executive work or to laboratory duties are entitled to be paid in money. This principle is rapidly gaining recognition; it is in accordance with it that salaries ranging from five hundred dollars to five thousand dollars per annum are now paid by the hospitals of New York to resident and non-resident pathologists and assistant pathologists. Similarly, we may note the addition of paid examining physicians to the staffs of Bellevue, St. Luke's, and other hospitals, and the payment of salaries to permanent resident physicians attached to the private wards of several hospitals in New York.

IV. THE MEDICAL STAFF IN RELATION TO THE RIGHTS OF ITS MEMBERS.

We have thus far treated the medical staff as an instrument for the accomplishment of certain ends for which the hospital chiefly exists. Before concluding it will be fair to ask, What are the rights of the men who form our medical organizations?

The Right to Remuneration. The question of salaries has already been dealt with in the treatment of hospital economy. While under the prevailing form of organization salaried members of the medical staff are few, so great are the advantages associated with hospital service that the average physician who gives only part of his time to the hospital considers himself amply repaid. The rule may be laid down that those whose opportunities for earning a livelihood outside of the hospital are directly promoted by their hospital connection may reasonably be expected to give their services to the charity patients of the hospital without money return; while those whose hospital work interferes with the acquisition of private practice are entitled to remuneration in proportion to the sacrifice made and the service rendered.

In building up a hospital organization the question of medical salaries must not be allowed to occupy too conspicuous a position. The form of organization adopted and the assignment of duties should have reference mainly to the functions of the hospital. If the proper treatment of patients requires the creation of positions which exact much and give nothing in return, salaries will find their proper place as the work of the hospital unfolds.

THE RIGHT TO COLLECT FEES FROM PRIVATE PATIENTS. Whether the attending and resident staff receive salaries or not, or whether some receive salaries while others do not, no payment of fees should be tolerated for the treatment of patients in the charity wards; but we must not permit well-to-do patients to enter the hospital for the purpose of escaping the payment of the legitimate fees of the physicians or surgeons whose services they seek.

Private wards exist for the convenience of the well-to-do classes, from whom it is right to demand suitable payment for everything furnished. The service given by the attending physician is distinct from that rendered by the hospital, and should be paid for at the rates current for such service and without deduction for the benefit of the institution. The daily or periodical charges of the hospital should fully cover its expenses, and there will then be no excuse for the exploitation of the staff by the institution.

THE RIGHT TO PUBLIC RECOGNITION. There should be due recognition, in annual reports and other official publications, of the work done by the medical staff. All the sacrifices made for the hospital and its patients are not accounted for in the subscription lists. However just we may strive to be, however sure that our scheme of organization is such that the staff is properly compensated for everything it does, many medical men, inspired by the most praiseworthy motives, will continue to contribute to the hospital far more than can ever be repaid; to these honor should be ungrudgingly given.

PERMANENCY OF TENURE AND PROMOTION FOR MERIT. Not by public recognition alone should the hospital reward those who give their strength unreservedly to its cause. It should guarantee permanency of tenure to those whose duties are faithfully performed, permitting them to hold office without interruption so long as they are physically and mentally capable of meeting the demands of active service, promoting them from dispensary to hospital, from positions of small opportunity and scant emolument to places of greater responsibility and wider influence; and when their fitness for strenuous professional life is terminated by advancing infirmity, the hospital should elevate them to honorary positions which will advertise to the new generation the work that has been done and the distinction earned.

The Right to Intellectual and Professional Development. Since the hospital is the scene of the chief activities of many of the best minds in the medical profession, is it not right that it should give to the intellectual powers of its staff the freest play, consistent with the good of its patients? If this is done there will be provided proper facilities for a reasonable amount of scientific research; protection from the enforced superficiality which results from the assignment of too many patients to one man; compulsory vacations to protect the self-forgetful worker from the consequences of his own zeal, and to afford opportunities for the development and training of assistants on whom larger responsibilities are ultimately to fall; permission to teach and to profit by contact with younger men, who bring with them from college new problems and the revelations of a constantly unfolding science.

Participation in the Councils of the Hospital. We must not shirk the question of the right of the medical staff to participate in the hospital councils. The vital question is not whether the governing body of the hospital consists exclusively of laymen or whether it is a joint body of laymen and physicians; for in any event, from the very nature of the hospital's work, medical opinion is bound to play a large part in formulating its policies. Even when absolute power of appointment and of removal of members of the medical staff is vested in the lay board, the medical board will exercise a powerful influence if the members of the medical staff are wise, honest, well qualified for their duties, and loyal to the hospital and to one another. No body of hospital trustees can escape the influence of a medical board so constituted, and no board of trustees that is worthy of its trust wishes to do so. I believe, therefore, that the so-called "fight for medical representation in hospital management" is a needless fight.[10]

It is best to leave to the lay board without question all the power and all the responsibility of initiating hospital enterprises, of fixing the budget, of securing public or private support, of appointing executive officers, of receiving and disposing of legacies and donations. In matters that are purely medical or in which the professional qualifications of medical men are concerned, the medical staff is entitled not only to exercise its moral influence, but to have representation and voice. I know of no better plan to secure this than that which has long been in operation at Mt. Sinai Hospital, where a standing committee of the board of lay directors, and a committee of like number appointed by the medical board, are united in a conference committee. This committee meets at the call either of the president of the hospital or of the president of the medical board; and all medical matters affecting the interests of the hospital are referred to it for consideration and report before final action is taken.

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[10] A. A. Eshner, M.D., Medical Representation in Hospital Management, Phila. Med. Jour., September 20, 1902.

The rights of the medical staff are as few and as simple as their duties are many and involved. But few as they are, their proper recognition is an indispensable condition of healthy hospital organization. To uphold them there is needed not so much a militant medical board, with a conspicuous chip on its shoulder, as an honest acceptance of duty, sympathy with the aims of the institution, and a self-respect which scorns professional jealousy. In the hands of a medical board which stands ready to promote talent, to reward industry, and to labor devotedly for the welfare of the hospital, the rights of the medical staff are secure.

ABNORMAL INVOLUTION OF THE MAMMARY GLAND, WITH ITS TREATMENT BY OPERATION.

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The changes which take place in the mammary gland of the female during the declining stage of its life history are very radical, and lead eventually to an almost total disappearance of the gland tissue, including both ducts and acini. It is during the period of this profound change that we meet with the condition commonly known as "cystic disease" of the breast. Coming, as it does, at a period of life when cancer is most frequent, and presenting symptoms which it is often most difficult to distinguish from those of that much-dreaded disease, this affection assumes a clinical importance which has long been recognized by both patient and physician.

To the lay mind a tumor of the breast is so suggestive of cancer that the patient is quite ready to submit to any measure, however radical, for its relief. Uncertainty as to the true nature of cysts of the breast has given rise in the professional mind to a diversity of opinions. By one a cyst is considered a harmless lesion which may be let alone, and this advice is often given, as the physician fears to place the patient in the hands of a surgeon who is inclined to amputate, as a routine treatment, every breast, whether it be the seat of a cyst or a malignant growth.

The true position of mammary cysts in the catalogue of morbid processes becomes, therefore, one of great importance from a practical point of view. Cystic disease of the breast has long been the object of careful study both by the surgeon and the pathologist. Opinions seem to have been divided as to whether cysts are formed in the breast by obstruction solely, as was the view of the older pathologists, or whether an active new-growth of acinal epithelium was an essential factor in their development. Since Réclus, in 1887, called attention more especially to the epithelial origin of these structures, many different theories have been advanced to explain the occurrence of these multiple cysts in the involuting breast.

In a paper read before the American Medical Association,[1] I have endeavored to classify these theories into three main groups. The first of these may be described as the inflammatory theory, the second the new-growth theory, and the third the involution theory. König, in his paper on chronic cystic mastitis, is a strong advocate of the inflammatory origin of this condition, a view also held by many French and English surgeons. The chief points on which the inflammatory theory is based are the evidences of chronic inflammation in the round-cell infiltration and thickened stroma of the gland. The clinical course of the affection is also thought to point to its inflammatory origin, and stress is laid (König) on the swelling and tenderness of the breast, particularly at the catamenial period, and on the tendency to frequent exacerbations so characteristic of chronic inflammatory processes.

The new-growth theory is advanced principally by German observers, who see in the very marked changes in the epithelium lining the cysts and the neighboring acini evidence of neoplastic formation. Ordinarily these cysts are lined with a single layer of epithelium, and the thin wall is typical of a retention cyst. In other cases there is a remarkable activity in this lining membrane, so that the epithelial cells become heaped up and assume a gland-like structure. A microscopic study solely of such specimens may readily give rise to the impression that one has to do with an active neoplasm of an epithelial character. This, however, is not by any means a constant condition, and the tendency to epithelial proliferation cannot be regarded as the sole characteristic feature in the development of cysts of the breast.

A later theory still is based upon a study of the process of normal involution of the breast, and presents a more rational solution of their development. As the menopause approaches, the mammary gland enters into the declining stage of its life history. Palpation of the breast at this period (between forty and fifty years) gives a nodular or shot-like sensation to the fingers which is most characteristic. This is due to a disappearance of the acinous tissue of the

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[1] Jour. Amer. Med. Assoc., July 15, 1905.

gland, and to an increase in the connective tissue. Many ducts and acini may become obstructed during this process, and a dilatation ensues which results in the formation of minute cysts. To this nodular condition of the gland I have applied the term "cobblestone"—the nodules, consisting of slightly dilated ducts or acini, surrounded with a thickened connective-tissue stroma, lying close to one another, and making up the substance of one or more segments of the gland.

Up to this point the process may be considered physiological. Any exaggeration of these processes may result in more marked cyst-formation at some one point and the production of a distinct tumor in the breast, and the condition known as "abnormal involution" exists. On laying open such a breast, usually one or two larger cysts are found, and many small cysts are seen dotted over the different segments of the gland. The process is as a rule diffuse, although one lobule is often noted as the situation in which the disease is most advanced. The cysts are filled with a dark-colored fluid, and before opening present a greenish tint in contrast with the dull-white tissue of the gland, which shows a condition of marked fibrous induration. The color of the contained fluid may vary through all shades of brown, green, and black.

The age at which these cysts are more frequently found is included, in my own experience, between the forty and forty-five year limit. On examination by palpation a tumor is felt in the upper and outer quadrant more frequently than elsewhere, although any of the quadrants may be the seat of this affection. In such case the other quadrants of the breast, and the other breast also, are found to present a cobblestone sensation to the palmar surfaces of the fingers. Pain and tenderness are present in about one-half of the cases, and these symptoms are more obvious at the catamenial period. Enlarged axillary glands are detected in only a small proportion of the cases. It has been my impression, based upon clinical experience, that inversion (not retraction) of the nipple is a frequent accompaniment of abnormal involution, and it may certainly be stated that previous diseases or abnormalities of the breast, especially those which interfere with the normal function of the gland, are predisposing factors. Single cysts of considerable size are occasionally found as the result of obstruction of one of the larger ducts, caused either by a plugging of the lumen or by the contraction of a cicatrix.

There is a marked difference in the lining membrane of these cysts and one which has a distinct pathological import. In the paper already referred to, 758 cases of tumors of the breast were analyzed. Of these 115 (15 per cent.) were cases of abnormal involution. Of the 115 cases of abnormal involution, 59 showed conditions which may be classified as cystic only; that is, there existed cysts lined with a single layer of epithelium in what may be called a quiescent state

(Fig. 1, and Fig. 5, *A*). The fibrous thickening of the stroma of the gland occurring during the involution process had, in these 59 cases, produced by obstruction a dilatation of the ducts, without obvious changes in the character of the epithelium. In the remaining 56 cases the epithelial cells contained within the cysts showed evidence of a tendency to undergo proliferation. Thus, nearly half of the cases presented conditions of activity of the epithelial structures. I have, therefore, divided the cases of abnormal involution into two distinct groups—the cystic group and the proliferative group.

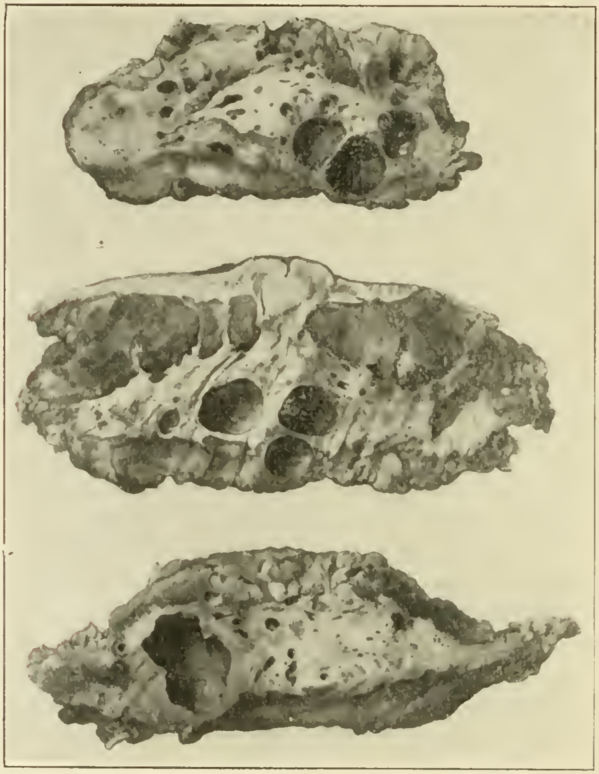

Fig. 1.—Abnormal involution, cystic type. Three parallel sections from the same breast showing the extensive involvement of the gland. The central section is made through the nipple. Under the skin is considerable subcutaneous fat, and beneath this is seen the gland tissue which reaches the surface at the nipple. The ligaments of Sir Astley Cooper connecting the breast with the skin are plainly seen. The breast is riddled with cysts varying up to 1.5 cm. in diameter, which were filled with clear straw-colored fluid. Practically no normal gland tissue is found, the breast consisting of cysts separated by a small amount of fat and considerable dense fibrous tissue infiltrated with small round cells. Microscopically, there was no epithelial proliferation, the cysts being lined by a single layer of flattened cells. The few ducts and acini found in the fibrous tissue were small and distorted.

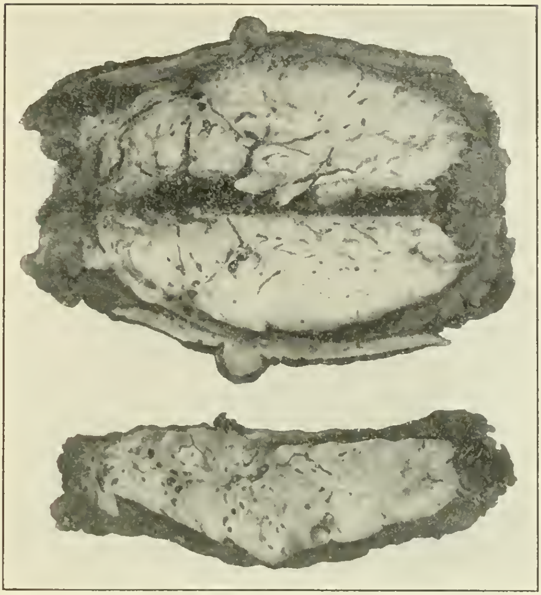

FIG. 2.—Abnormal involution with some cyst formation and epithelial hyperplasia. Two sections from the same breast showing a large amount of dense, dull, white breast tissue. The breast contained numerous cysts, most of which were small, one, however, being 3 cm. in diameter. These were filled with opaque greenish fluid. The cut surface showed many minute translucent points representing acini, and microscopically there was considerable epithelial hyperplasia. The other breast (Fig. 3) was similarly affected, but the cyst formation was not quite as marked.

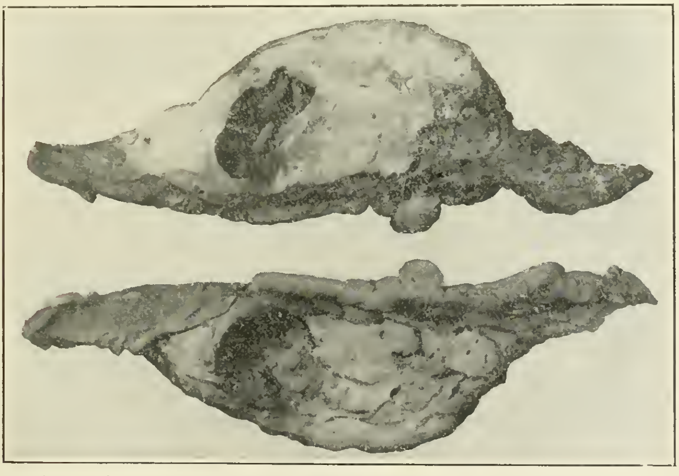

FIG. 3.—Abnormal involution with cyst formation and epithelial hyperplasia. The specimen is from the same patient as Fig. 2, and shows the condition of the other breast. Microscopic examination of the breast showed some increase in the number of acini; the ducts were somewhat dilated and the epithelium lining them was heaped up, forming in some papillary projections, and in others completely filling the lumen.

Abnormal Involution.
- I. Cystic group.
- II. Proliferative group.
  - (*a*) Acinal form.
  - (*b*) Papillary form.
  - (*c*) Adenomatous form.

Although it is possible thus to divide cases of abnormal involution into several groups according to the pathological changes present in them, it should be stated that clinically we find no characteristic which enables us to distinguish these forms from one another.

This brings me to the principal purport of this paper, which is to call attention to the more or less active epithelial processes which are going on in nearly half of the cases of abnormal involution.

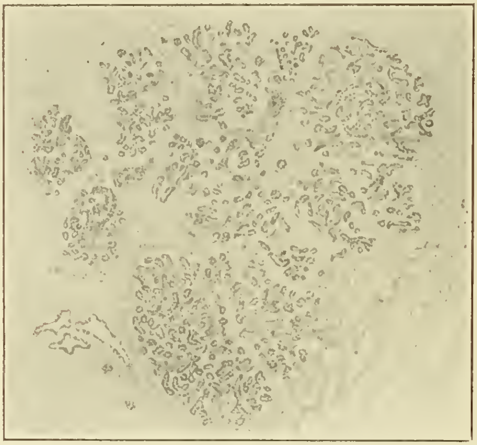

Fig. 4.—Abnormal involution, acinal hyperplasia. The acini are actually increased in number as compared to what is usually found in a quiescent breast from a woman of the same age. Other sections from the same breast showed cystic changes, but epithelial changes—other than the acinal type—were not marked.

Of the proliferative group of cysts we may distinguish three subdivisions, according to the character or degree of the epithelial growth: (1) Proliferation of the acini, (2) papillary outgrowth of the epithelium into cysts, and (3) adenomatous proliferation of the epithelium. Of this group of 56 cases. 36 were hospital cases, and the data preserved made possible a careful microscopic analysis of the varying conditions of the epithelium. Of these, 10 came under the first group (Figs. 2, 3, and 4). Here there was an apparent increase in the number of gland acini when compared with normal breasts of the same age, and conditions of marriage, childbirth, and lactation. This increase in the number of acini was accompanied in the majority of instances by a proliferation of the epithelium to such an extent that thickened or even solid columns of cells were produced, retaining, however, the characteristic formation of gland ducts and presenting no infiltration beyond the basement membrane. Although this group gives no direct evidence bearing upon the possibility of a transition from involution to carcinoma, it should be stated here that in 3 cases of cancer of the breast occurring in the above series of 758 cases of tumors of the breast, this form of abnormal involution was also observed.

The second class of epithelial proliferation observed was one which may be described as papillary proliferation (Figs. 2 and 5). By this term is meant a growth of epithelium in the cyst cavities of such a nature that the epithelial cells become heaped up and project into the cavity without a connective-tissue pedicle or support. The microscopic picture of this condition suggests that more epithelium is produced on the cyst wall than can be accommodated on the basement membrane, with the result that certain portions are forced out toward the centre of the cavity. There were 18 cases of this type of abnormal involution.

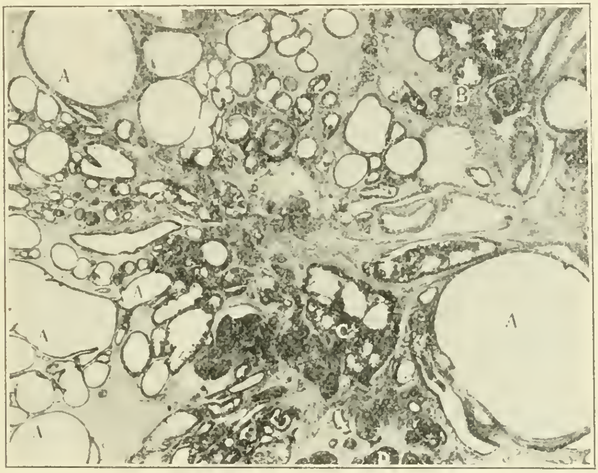

Fig. 5.—Abnormal involution with marked epithelial hyperplasia and adenocarcinoma. (Low power photomicrograph.) The section shows very extensive cyst formation and the papillary and adenomatous types of epithelial change. Most of the cysts, particularly the larger ones, are lined with a single layer of epithelium. Many of the small cysts are filled with solid masses of epithelial cells, which in places have a tendency to form atypical glands. Where the proliferation is not as marked the cells are heaped one upon another, forming papillary ingrowths. In places the epithelium has broken through the basement membrane and is infiltrating the surrounding tissue. This infiltration is not well seen on account of the low magnification. *A*, simple cysts; *B*, papillary epithelial projection; *C*, adenomatous arrangement of proliferated epithelium; *D*, adenocarcinoma.

The third and last type of epithelial proliferation is that to which Tietze and Greenough and Hartwell have applied the name of "adenomatous" proliferation (Figs. 5 and 6). This condition must be regarded as a somewhat more advanced type of epithelial growth, one in which the papillary outgrowths from different portions of the cyst wall appear to meet and fuse together. The resulting microscopic picture is that of a space filled with epithelial cells, in which here and there an open gland lumen is observed. This form of proliferation occurred in 8 of the hospital cases and in 4 of the cases from private practice. This variety has a special interest from the fact that it is precisely under these conditions that we most frequently find the combination of abnormal involution and carcinoma. The transition stage is observed when the epithelium no longer confines itself to the cyst cavity, but breaks through the limiting membrane and infiltrates the adjacent structures. In such microscopic specimens cyst formation and carcinoma may be seen at either end of the field with the intervening transition stage (Figs. 5 and 6).

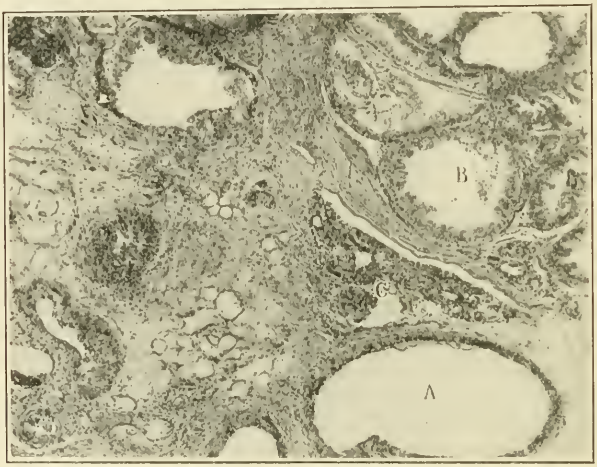

FIG. 6.—Abnormal involution with extensive epithelial hyperplasia and adenocarcinoma. (Photomicrograph from the same section as Fig. 5, higher power.) The section shows in one portion a simple cyst lined with somewhat flattened epithelium. In the other cysts the epithelial overgrowth is more marked, and papillary projections are well shown. Near the centre of the section is an area of adenocarcinoma. *A*, simple cyst; *B*, cyst with papillary projections; *C*, adenocarcinoma.

In 9 cases of cancer of the adenocarcinoma type and in 3 cases of the medullary type there were associated involution changes of a papillary and adenomatous character. We find, therefore, that in a collection of 115 cases of abnormal involution, 15 cases of this disease in combination with carcinoma were obtained, or 13 per cent. This figure closely approaches the actual condition, but is somewhat greater than the 10 per cent. found by Tietze and Greenough and Hartwell.

Thus we see that in a certain proportion of cases we have the possibility of cancer to face, although the great majority of cysts of the breast are of a benign character. Spontaneous rupture of a large cyst is a not uncommon occurrence, the patient believing her tumor to have disappeared, and it is probable that quite a number of patients have obtained a spontaneous cure in this way, but the history of a large number of cases in my experience is that the cyst has reformed. Eventually the breast may become riddled with cysts of a large size, and the gland tissue totally disappear, both breasts often being affected. Occasionally suppuration is observed, but this is of rare occurrence. More frequently the disease becomes stationary at a certain stage of its development, but always remains as a menace and a source of anxiety to the patient.

The following cases are given as illustrating fairly well the different types of this affection. The hospital series (Cases I to IV) has been selected from the collection of cases already referred to; Cases V and VI are from private practice.

CASE I.—*Abnormal Involution with Cyst Formation and but Little Epithelial Change.* The patient, a widow, aged forty-eight years, had had no children. Ten years before presenting herself for treatment she received a blow on the left breast, and two months before, a tumor appeared at that point, and increased slowly in size, without pain. On examination, in the upper outer quadrant of the left breast was a hard, freely movable new-growth the size of an English walnut. It was not tender. The nipple was normal, and no axillary glands were noted. A quadrant of the breast was excised.

Pathological report (abstract): The specimen consisted of an irregular piece of breast tissue and fat 2 x 4 x 6 cm. in diameter. On section there was a large amount of dull-white breast tissue in which a few glistening points representing acini could be seen. In one place was an opened cyst 2 cm. in diameter. Microscopic examination showed a few acini separated by a large amount of fibrous tissue. The periductal fibrous tissue was also increased, and there was considerable small round-cell infiltration. The ducts were dilated, forming small cysts lined with a single layer of epithelium, and contained cholesterin crystals and detritus.

CASE II.—*Abnormal Involution with Acinal Hyperplasia and Some Epithelial Proliferation.* The patient, aged forty-nine years, was married, but had had no children. She had, at the time of examination, a tumor in the breast the size of a hen's egg, which she had noticed for six weeks. It was not painful or tender and was freely movable. The nipple was normal, and there were no glands in the axilla. A plastic resection was performed.

Pathological report (abstract): A piece of breast tissue was received which contained numerous cysts, varying up to 1.5 cm. in diameter, filled with thick, yellowish secretion. Microscopically there was an increase in the number of the acini, with dilatation of the ducts. The cysts thus formed were lined in general by a single layer of epithelium, while in some the epithelium was heaped up, forming cellular papillary ingrowths.

cancer is suspected it is well not to forget to palpate the abdomen in the hepatic region. I take the liberty of introducing these details, as the ordinary methods of examination are of a more or less superficial character, if the statements of patients are to be relied upon.

If the breast is in the declining stage of its life history the "cobblestone feel" will be observed in one or more quadrants, more often the upper and outer quadrant, and the surgeon must expect to find this quadrant enlarged upward and outward in the direction of the axilla. The mammary gland is often described as a trefoil in shape. The projection of the margins at certain points which this shape implies is, however, most marked in the upper and outer quadrant, so that the term "pear shape" would better describe the ordinary outline of the organ. Accompanying this condition of the gland is often an increased sensitiveness, particularly in those parts where this normal involution process is most marked.

If a cyst is present it will readily be distinguished from the surrounding gland tissue, but the examiner must not expect to detect fluctuation. Such tumors are often externally hard to the touch, and this is due to the tension of the cyst walls. The outline, as a rule, is better defined than that of a cancer nodule. As both cancers and cysts are more frequently found in the upper outer quadrant, the situation of the nodules does not throw any light upon the diagnosis.

The absence of enlarged glands in the axilla is, however, a great encouragement to hope for the absence of cancer. The presence of marked signs of involution, with the absence of glands in the axilla, is a symptom group strongly suggestive of the presence of cystic changes of a benign character.

The average age of the 41 patients already referred to was forty-four years at the time of presenting themselves for treatment. It is noticeable that there was comparatively little variation in the age recorded—3 patients only being over fifty years and 4 below forty years.

Treatment. The question has been raised whether this affection needs any surgical treatment. The diagnosis once satisfactorily established, many physicians may argue that surgical intervention is unnecessary, that in the fulness of time the growth of the cystic tumor will cease, and may spontaneously disappear. The patient also is easily persuaded that, inasmuch as the disease is not malignant, the question of operation may be indefinitely postponed.

It is certainly true that accidental rupture of the cyst wall may not infrequently lead to an obliteration of the sac, and aspiration of the cyst has, in the hands of the surgeon, been followed by an equally favorable prognosis. Why then interfere in so simple and easily remedied a malady? It has been the object of this paper to supply an adequate answer.

Bull, Abbé, McGraw, and others formerly advocated aspiration and evacuation of the cyst, and report cures. McGraw, however, reports 2 cases of cancer developing after this treatment. Rupture of the cyst by forcible massage has been recommended by Herbert Snow. A large thin-walled cyst might be as easily and perhaps as painlessly evacuated in this way as by puncture, but both methods are open to the objection that numerous cysts are left untouched, the prognosis of which no one can give with any certainty. A patient recently informed me that a small cyst of the breast had disappeared after massage by a nurse. Massage seems to me a most undesirable treatment for any affection of the breast. A complicated and highly sensitive glandular structure like the mammary gland, with its numerous long, slender, and branching tubes, is easily bruised and a lesion produced which may be the starting point of serious organic disease.

The objection to operative treatment has been based in the past upon the custom which has only too largely prevailed, of amputating the breast for every suspicious lump—the surgeon leaving with unconcern the final diagnosis to the pathologist. Secure in the belief that this in the long run is a safe policy, the operator is not disconcerted to find that he has removed the breast for a benign growth.

Amputation is an operation which every woman justly shrinks from, not only on account of its magnitude, but for the great disfigurement which it involves—nothing but the threat of cancer will drive her to it. Most women also object strongly to unsightly scars, the result of an exploratory incision directly over the tumor. As cysts are more frequently situated in the upper hemisphere, incisions over this region come within the area of a surface often exposed to view. It is true that puncture of the cyst does not involve any deformity, and that a single cyst may often be cured in this way. Puncture for exploratory purposes is now pretty generally considered inadvisable, owing to the danger of spreading the infection in case of cancer. It is a procedure which, formerly advocating for diagnostic purposes, I have now abandoned.

If now a patient presents herself with a tumor of her breast, what line of treatment should be proposed? It is obvious that the consultant should be able to satisfy the attending physician, as well as the patient, by an opinion which will definitely settle some future line of action. The time has now arrived when our knowledge of the diseases of the breast should enable us to say conclusively either that there is no occasion for operative interference, that there is no tumor (and it is astonishing how often the family physician, as well as the patient, is deceived), or that the indications for an operation clearly exist.

This task I find all the more easily performed since the adoption of the method of operating to which I have given the name "plastic resection of the mammary gland." This operation involves no scar visible to the patient, or other disfigurement of the breast, and cancer is suspected it is well not to forget to palpate the abdomen in the hepatic region. I take the liberty of introducing these details, as the ordinary methods of examination are of a more or less superficial character, if the statements of patients are to be relied upon.

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This task I find all the more easily performed since the adoption of the method of operating to which I have given the name "plastic resection of the mammary gland." This operation involves no scar visible to the patient, or other disfigurement of the breast, and is an operation from which the patient may be convalescent in a week's time. At the same time, the question of cancer is not only definitely disposed of, but the quadrant containing the larger cysts (which represent the tangible tumor) has been removed; the other quadrants have been explored, smaller cysts laid open, and the involution process speeded on its way to resolution of the gland tissue into simple connective-tissue elements.

The operation consists in an incision differing somewhat from the so-called Thomas incision, in that it lies principally along the outer border of the breast and extends from the lower end of the outer hemisphere along the axillary border, and is of sufficient length to expose freely the upper and outer quadrant of the breast, which is so often extended outward and upward toward the axilla. The incision being carried down to the pectoral fascia is continued on in the loose tissue between that fascia and the fascia of the mammary gland, while the other hand of the surgeon everts the anterior wall of the wound thus made and exposes freely the posterior wall of the gland. It is now easy to recognize the presence of the cyst with its slightly greenish-colored contents shining through the fascia.

The segment of the gland containing the larger cysts, generally one or two in number, should be carefully excised without opening them if possible. Although ordinarily their contents are aseptic, it is better to avoid contamination of the wound, especially if the cyst is near the nipple. This is accomplished by a V-shaped incision, the apex of the V lying directly under the nipple in the central portion of the gland. The remaining segments of the gland are now incised by radiating incisions to detect the presence of any smaller cysts. The number of these incisions may vary from two or three to six or eight. Any doubtful spot can thus be readily excised and submitted to a microscopic examination, which should be made at the time, and it is my custom always to have a pathologist present at the operation for this purpose.

It may be said here that this operation is the ideal one for exploration in doubtful cases. If the nodule is cut into and found to be cancer, the preliminary incision has already separated it from the axillary glands, and any danger of mechanical transference of portions of the primary lesion to distant parts is avoided. The incision into the nodule in this case may be closed by a suture while the operation for cancer is carried out. After the gland has been thoroughly explored by radiating incisions and one segment with the larger cyst secured, the next step in the operation is the closing of the wounds in the gland itself. The V-shaped space may be closed by a double row of sutures, so that the accurate adjustment of the sides of the V may be effected. This avoids any depression externally. The radiating incisions usually close spontaneously when the gland is laid back again upon the pectoral muscle. A stitch here and there may, however, be necessary to prevent any tendency to eversion.

The outer edge of the gland is stitched to the margin of the pectoralis in order to hold it securely in its normal position. For all buried sutures and ligatures catgut is the preferable material, as it is quickly absorbed and does not leave, like silk, a remnant behind to become a source of irritation and suspicion.

The outer wound is closed by silkworm-gut sutures. The somewhat puckered or dimpled appearance of the contour of the breast, which is at first noticeable, soon disappears as the buried sutures become absorbed, and the deeper portions of the wound adjust themselves to their new relations. The stitches can be removed within a week's time, and the patient can be promised that confinement to bed is to be of a few days' duration only. No drainage is used, and in all but 3 cases the wound has healed by first intention. In these the removal of a stitch or two for drainage was all that was necessary to produce prompt healing of the wound. In case of a large cyst, or one very near the nipple, being accidentally opened, I am in the habit of sponging the wound with a weak solution of corrosive sublimate.

It is astonishing to see how little change in the appearance of the breast is produced by the operation. The skin incision is invisible to the patient herself as she stands before the glass. At the end of two or three months careful palpation fails to reveal the site of the deep incision. It is only by reference to my notes that I am able to tell which portion of the gland has been removed. I have performed this operation for cysts of the breast in all fifty times, and have never failed to be able to make a diagnosis at the time of operation, and have never had any reports of a recurrence of cystic or other disease in the patients operated upon.

Surely an operation which is comparatively a light ordeal to the patient, which removes all doubt as to the nature of the tumor, and all probability of a secondary development of cancer, is one that should commend itself above all others. A greater familiarity upon the part of practising surgeons with the classification of tumors of this organ, and with the gross pathological appearances, would doubtless save many a breast from being amputated.

THE SURGICAL ASPECTS OF GASTRIC CARCINOMA.[1]

By John B. Deaver, M.D.,

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Cancer of the stomach is a disease of frequent occurrence. From 25 to 40 per cent. of all cancers in the body are primary in the stomach. Statistics[2] show that in Baltimore there are over 200 deaths annually from cancer; in Boston there are over 300, in Philadelphia nearly 500, and in New York nearly 900 deaths every year from some form of cancer. Of these deaths more than a fourth, and perhaps almost a half, are directly due to cancer of the stomach. Dowd[3] says that the United States Census Report for 1900 recorded 9000 deaths from cancer during that year, that 31 per cent. of fatal cancers were in the stomach, that probably many cancers of the liver and the abdomen were primarily gastric, and that many cases of gastritis and allied diseases were really carcinomatous in nature.

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[1] Read at the meeting of the Southern Surgical and Gynecological Association, Baltimore, December 11, 12, and 13, 1906.

Medical measures are powerless to cure cancer in any region of the body. Escharotic pastes, cauterizing applications, detergent washes, serums and antitoxins, and even the subtile power of the *x*-rays and the Finsen light are all of them powerless to cure external cancer. How much more improbable it is that any save surgical remedies will cure cancer of the internal organs! That, in the case of inoperable tumors, medical measures will sometimes prolong life and mitigate suffering, no one would be so foolish as to deny. The wide experience of Jacobi,[4] so cautiously and conservatively expressed, in regard to the beneficial effects of methylene blue (methylthionin hydrochloride) in such patients, is a gratifying example of what may be done by medical science even for hopeless cases. But, as Bland Sutton[5] said a number of years ago, as long as we are ignorant of the cause of cancer, so long must the only successful treatment be the extirpation of the growth; and I may add that the sooner this is done the better for the welfare of the patient and for the reputation of the family physician and the surgeon.

If it were possible to prevent cancer, what then? Before answering this question one would have to determine the cause of cancer; and of its specific cause we are absolutely ignorant. That it is caused by some form of micro-organismal life it seems safe to infer; but beyond such an inference even the most enthusiastic pathologists will not go.[6] But we do know many of the predisposing causes of cancer, and especially well recognized are the predisposing causes of gastric cancer, which is all that concerns us now.

Mumford and Stone[7] traced 60 patients who had been treated at the Massachusetts General Hospital for chronic indigestion, and who subsequently died. Of these, no less than half died of gastric carcinoma. They further made a study of 50 patients with gastric carcinoma, and learned that in no less than 41 of the 50 patients there was "a history either of ulcer or of long-continued digestive disturbance, of which the exact nature could not be ascertained." Mayo[8] found a history of ulcer or other disease in 36 per cent. of his patients with cancer of the stomach, and clear evidence of cancer developed on ulcer in 30 per cent. of the last 40 pylorectomies and partial gastrectomies. Moynihan[9] noted a history of gastric ulcer or chronic indigestion in 27 out of 45 patients with cancer of the stomach (60 per cent.). Among 22 patients with gastric carcinoma, so far advanced as to be operable only by gastroenterostomy, no less than 16, or over 72 per cent., gave such a history. Jedlicka[10] quotes Ssapesko as stating that among 100 cases of gastric carcinoma only 10 were found in which the cancer was not engrafted on a preceding ulcer. Further statistics could no doubt be quoted; they would merely serve to emphasize the point that I am trying to make, *that chronic gastric dyspepsia is the chief predisposing cause of cancer of the stomach.*

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[2] Bryant, quoted by Van Valzah and Nisbet, Diseases of the Stomach, London, 1900, 513.

[3] N. Y. Med. Record, 1906, i, 91.

[4] Jour. Amer Med. Assoc., 1906, xlvii, 1545.

[5] Tumors, London, 1901, preface.

[6] See Gaylord, American Practice of Surgery, edited by Bryant and Buck, New York, 1906, i, 403.

[7] Surgical Aspects of Digestive Disorders, New York, 1905, pp. 87 and 201.

We know that cancer of the stomach presents clinically two forms. In one, a patient past middle life, without having suffered previously from indigestion, suddenly loses appetite especially for meats, grows progressively weaker and more emaciated, develops epigastric pain and possibly a palpable mass, becomes subject to vomiting spells every few days, which bring up a mass of coffee-ground material, foul smelling and fermented, and quickly develops the cancerous cachexia. This is the classical picture of gastric carcinoma, and it is still seen; but it is of rarity when compared with that other course of development which we have only begun to appreciate in the last few years. This second and much larger course is found in patients who have been life-long sufferers from dyspepsia. Medical treatment has at times given relief, but the indigestion recurs again and again. Appetite may be preserved, but digestion is torture; so abstemiousness becomes second nature. There may or there may not have been some period when gastric ulcer was suspected or actually diagnosticated. Usually there have been no very definite symptoms, and the patient has been treated for chronic gastritis. Finally these patients die, worn out with suffering, with prolonged starvation, and wearied with ineffectual drugging. At the autopsy a wide-spreading epitheliomatous ulcer is found, distorting the stomach, stenosing the pylorus, and binding the colon, the pancreas, the stomach, and perhaps the liver and the gall-bladder together in an indistinguishable mass of adhesions. The wonder is that such patients live so long.

Experience in the surgery of the upper abdomen within the last few years has taught surgeons much, but they have learned nothing of more importance than the security and certainty with which mechanical defects in the physiological action of the stomach may be remedied by mechanical means. If chronic and rebellious cases of indigestion were promptly turned over to the surgeon, and if suitable operations were done on such patients, there would be fewer cases of carcinoma of the stomach observed by physicians. Every case diagnosticated certainly as carcinoma of the stomach before operation is a disgrace to the attending physician, provided he has had the patient under treatment for more than a few weeks. Patients who present symptoms of gastric indigestion which will not yield in that time to medical treatment should be subjected to operation; and if operation is not undertaken, and the patient's disease is allowed to continue uncured until a diagnosis of cancer has been made, the disgrace and the responsibility rest on the attending physician. We know beyond peradventure that such patients as these can be cured by operation, and that the development of carcinoma may be thus prevented. For physicians not to recognize this fact and act accordingly is not only unjustifiable, it is criminal.

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[8] Jour. Amer. Med. Assoc., 1905, ii, 1211.

[9] Trans. Clin. Soc. Lond., 1906, xxxix, 84.

[10] Operat. Behandl. d. Chron. Magengeschwürs, Prag., 1904, 24.

The point is now reached where it will, I think, prove beneficial to compare the results of operations on the stomach for benign disease with those for carcinoma of this organ. It is of interest first to know the immediate operative mortality, and then the proportion of ultimate recoveries or cures. In considering questions such as these it is injudicious to collect isolated operations reported at random by different surgeons. Statistics of abdominal operations which are so frequently done as are nowadays operations on the stomach present, when thus collected, a picture which is too likely to be distorted one way or the other: we obtain results which are neither phenomenally good nor phenomenally bad, but at any rate results which are far from the truth. In the earlier history of an operation such figures are of value, because the cases are few, and progress can only come when a sufficient number of observations has been collected for surgeons to study the principles which underlie the operation in question. Thus twenty years ago it was important to record every individual case of gastroenterostomy or pylorectomy, because no individual surgeon had at that time had the opportunity personally of observing the effects of more than one or two such operations. But now, when the experience of one surgeon may embrace a hundred or more such cases, the deductions to be drawn from such a series far outweigh in value those which were available even one decade ago from a series of similar size collected from various sources.

Let us consider, then, first the figures of gastroenterostomy for benign disease, an operation which may, I think, justly be considered as preventive of cancer. We have reports of large series of cases of this operation at the hands of various surgeons:

GASTROENTEROSTOMY FOR BENIGN DISEASE.

Operator.	Cases.	Mortality. Per cent.
Robson and Moynihan[11]	218	3.2
Mayo[12]	81	1.23
Czerny[13]	83	4.8
Mayo Robson[14]	97	1.03
Moynihan[15]	171	1.00
Deaver[16]	40	2.5
Morison[17]	27	3.7
Hartmann[18]	47	6.3

Now, it is a well-recognized fact that about 95 per cent. of patients who survive operation for benign disease remain well permanently. Consider these figures:

END RESULTS AFTER GASTROENTEROSTOMY FOR BENIGN DISEASES OF THE STOMACH.

Operator.	Cases traced.	Permanently cured.	
Barling	7	7	100.0 per cent.
Mayo	28	27	96.4 "
Moynihan	29	29	100.0 "
Czerny	53	44	83.0 "
Robson and Moynihan	20	20	100.0 "
Paterson (collective statistics)	116	109	93.9 "
Deaver	30	30	100.0 "
Robson	96	89	92.7 "

NOTE.—The latest statistics of Mayo in gastroenterostomy for benign disease give a mortality of practically 1 per cent.: actually 1.04 per cent.—2 deaths in 192 cases. My latest statistics show a mortality of less than 2 per cent.

May we not justly conclude that by timely resort to this operation many patients have been saved from the development of gastric carcinoma?

The cure of carcinoma of the stomach, when this dread disease has once developed, demands excision of the growth with the infected glands. The immediate operative mortality of partial gastrectomy is still very much higher than is that of the operation I have been considering, though the death rate has been reduced very much within the last few years.

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[11] Surg. Treat. Diseases of Stomach, New York, 1904, p. 177.

[12] Jour. Amer. Med. Assoc., 1905, ii, 1211.

[13] Petersen u. Machol, Beitr. z. klin. Chir., 1902, xxxiii, 297.

[14] Brit. Med. Jour., 1905, ii, 762.

[15] Lancet, 1905, ii, 362.

[16] Therapeutic Gazette, January 15, 1906.

[17] Brit. Med. Jour., 1905, ii, 777.

[18] Ibid., 772.

Partial Gastrectocy for Carcinoma.

Operator.	Cases.	Deaths.	Mortality. Per cent.
Mayo[19]	81 (25)	12 (1)	14.15 (4)
Kocher[20]	110 (58)	27 (9)	24.5 (15)
Mikulicz[21]	100	37	37.0
Mayd[22]	25	4	16.0
Krönlein[23]	50	14	28.0
Roux[24]	39	13	33.0
Meyer[25]	5	1	20.0
Kappeler[26]	30	8	26.6
Garré[27]	26	7	26.9
Hartmann[28]	22	7	31.8
Moynihan[29]	9	2	22.2
Paterson[30]	4	1	25.0

Note.—Mayo's latest statistics, as given in a personal communication, show a mortality of 11.45 per cent. for partial gastrectomy; while my latest statistics show a mortality of 11.11 per cent.

Taking, then, the more favorable recent figures of Mayo (4 per cent.) and of Kocher (15 per cent.), which offset to a certain extent the rather ancient statistics of Mikulicz, Krönlein, Roux, Hartmann, and others, we have a total of 393 partial gastrectomies with 104 deaths, or a mortality of 26.5 per cent., as the immediate result of operations for the radical cure of gastric carcinoma. This, it will be remembered, is to be contrasted with an immediate mortality of about 5 per cent. after operations for the prevention of this disease.

It will next be proper to determine what proportion of the 74 per cent. of patients who survive gastrectomy may reasonably hope to be ultimately and permanently cured of their disease. Let it not be forgotten that about 95 per cent. of patients surviving the prophylactic operation have been proved to be permanently cured. This question of the ultimate results of gastrectomy has been carefully studied by Moynihan,[31] who bases his conclusions largely on the statistics of Krönlein and Mikulicz, who alone of many operators have published in detail the terminal history of those patients whom they were able to trace. It is not the place here to discuss these statistics minutely, but it may be said that they show that patients suffering with gastric carcinoma have, under medical treatment, an expectation of life of about twelve months from the beginning of the disease. Many writers put it at less. Not only is this the duration of life, but it should not be forgotten that at the end of that time the patients under medical treatment will *all be dead.*

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[19] N. Y. State Jour. of Med., 1906, vi, 63.

[20] Paterson's Gastric Surgery, New York, 1906, p. 104.

[21] Paterson, loc. cit., p. 104.

[22] Paterson, loc. cit.

[23] Paterson, loc. cit.

[24] Paterson, loc. cit.

[25] N. Y. State Jour. of Med., 1906, vi, 68.

[26] Paterson, loc. cit.

[27] Paterson, loc. cit.

[28] Paterson, loc. cit.

[29] Trans. Clin. Soc. London, 1906, xxxix, 84.

[30] Gastric Surgery, New York, 1906, p. 178.

[31] Abdominal Operations, Philadelphia, 1905, p. 223.

There will not be even one who at the conclusion of that period will have had up to that time no recurrence of the growth, and who may, therefore, still be considered as curable—as potentially cured. If then surgery can show any permanent cures, and if the average duration of life under surgical treatment is longer, or at least not less than that under medical treatment, the conclusion is surely justified that surgical intervention is best for these patients.

To compare with an expectation of life of about twelve months for patients who have not been operated on, we know from Moynihan's studies that the average duration of life in patients who underwent gastrectomy in the clinics of Krönlein and Mikulicz was, from beginning to end of the disease, as much as twenty-four to twenty-six months—more than twice as long as the average duration of life without operation. Paterson's figures[32] from collective statistics give the duration of life after operation (86 operations in all) as an average of nineteen months after total gastrectomy (17 patients), of twenty-two and a half months after subtotal gastrectomy (14 patients), and of just over two years after partial gastrectomy (55 patients). It should be noted that Moynihan's figures refer to duration of life from the first symptoms of gastric cancer, while Paterson's refer only to the duration of life after operation. With this allowance, it is seen that the figures agree very closely; and as they are gathered from very different sources (Moynihan's from the German clinics and Paterson's largely from British sources), each series serves to confirm the other.

Not only, moreover, may the mere addition to the patient's life be of utmost importance from a social, commercial, or financial point of view, but death, when it does come, will attack the sufferer in a less hideous form. The patient will not die of starvation, as he would have done had no operation been performed. And although it would be heartless in the surgeon to tell his patient, when urging operation, that although the chance of permanent cure is slight, it is nevertheless altogether likely that he will die from cancer of the liver, with its attendent cachexia, and not from progressive starvation—although, I say, to draw such a picture of the future for his unfortunate patient would be diabolically cold-blooded in the surgeon, yet I doubt not that many a patient, dying of the cancerous cachexia some two years after the operation, will daily bless the art of surgery which has so prolonged his life and mitigated his suffering during the gradual approach of death.

But, though the chances of permanent cure are slight, they are not altogether imaginary. Among more than 100 cases of gastrectomy for carcinoma, Kocher[33] has had permanent cure result in 8 patients, who were reported as living and well at periods varying from three to sixteen years after the operation. Paterson[34] has collected, mainly from British sources, the end results of 79 cases of gastrectomy, 46 patients (58.4 per cent.) dying of recurrence within three years, and 33 patients (41.6 per cent.) being alive and well at the end of three years or more after the operation. No less than 12 patients (15 per cent.) were in perfect health more than five years after the operation, some being in good health at periods varying from six to fourteen years after the operation. We may expect, then, about 10 or 15 per cent. of patients treated by gastrectomy to be permanently cured without liability of recurrence.

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[32] Gastric Surgery, New York, 1906, pp. 106 and 107.

[33] Revue de gyn. et de chir. abd., 1906, x, 103.

Paterson's Statistics of End Results of Gastrectomy.

	Recovered	Traced.	Died since operation.	Still living and well.	
				Three years after operation.	Five years after operation.
Total gastrectomy	17	14	5	6	3
Subtotal gastrectomy	14	12	6	3	3
Partial gastrectomy	55	53	35	12	6
	86	79	46	21	12

There is another question of importance in regard to the surgery of gastric carcinoma which cannot be answered offhand. This is as to the advisability of excising ulcers or of doing a pylorectomy or a partial gastrectomy in the presence of benign disease as a preventive of malignant changes. I have argued in favor of gastroenterostomy as a prophylactic operation; and it may have been cause for surprise that I have not hitherto even mentioned excision of the ulcer or of the diseased gastric area. This ommission, however, has been intentional, since I desire first to contrast the immediate operative mortality of the two forms of surgical treatment. And in doing this I am, of course, not ignorant of the fact that gastrectomy in a cancerous patient is unavoidably a more fatal operation than it is in one suffering only from benign disease. But although I am quite willing to admit this fact, yet the operation of partial gastrectomy presents *per se*, and apart from the vitality of the individual patient, certain added risks, which in the present state of surgery are unavoidable, but which must, I venture to think, be obviated before the conscientious surgeon can recommend this operation in preference to gastroenterostomy in patients with benign gastric disease. The time may come, indeed I hope it is not far distant, when the operative mortality of gastrectomy will be reduced from 25 per cent., its present figure, to less than 10 per cent., but until that day has been reached I for one cannot feel justified in resorting to the more dangerous operation in cases certainly benign. I say this, too, with full knowledge and personal experience of patients who recover as blithely from a gastrectomy as they do from an interval operation for appendicitis. In my own experience with gastrectomy, comparatively small though it be, I have been surprised at the freedom from symptoms and the easy convalescence of some patients on whom quite extensive operations have been done; but, although increasing experience with the operation has made me regard it as neither so difficult nor so serious an undertaking as I formerly thought it, yet I have not yet reached, and I humbly trust never shall reach, that point where familiarity will breed contempt.

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[34] Gastric Surgery, New York, 1906, p. 108.

The arguments advanced in favor of excision of the ulcer or of the ulcer-bearing area, sound as some of them undoubtedly are, do not, in my opinion, outweigh the arguments against this treatment, which I have already stated. Though I firmly believe that the chief predisposing cause of gastric carcinoma is long continued dyspepsia due to ulcer or other form of benign gastric disease, yet I do not altogether credit the reports which would make us believe that about 1 out of every 10 ulcer cases is already cancerous.

Originally advocated by Rydgier,[35] who has recently[36] had the satisfaction of reporting his first patient as alive and well twenty-five years after the operation, pylorectomy was, nevertheless, a method which quickly fell into disrepute, owing to its enormous mortality. But in the last few years excision of the ulcer or of the pyloric region has again become popular with some surgeons (Maydl, Jedlicka, Ali, Krogius, Rodman, and others), chiefly as a prophylactic against the development of carcinoma, though there are also other reasons (absence of subsequent perforation or hemorrhage) which they urge for its more general adoption. Yet it seems to me that a diagnosis of "carcinomatous degeneration," based on microscopic findings, as to whose interpretation pathologists themselves are not unanimous, is at least as liable to be in error as is a clinical diagnosis of malignant disease based on the naked-eye appearance of a pyloric tumor found at operation; and it seems fair to conclude that patients in the former class might be as thoroughly cured by a palliative operation as have been those patients, by no means unknown to science, who presented a gastric tumor which was considered a naked-eye evidence of malignant disease too far advanced to be treated by excision.[37]

It is, therefore, with caution that I accept the conclusions of Jedlicka,[38] who argues very positively in favor of excision of benign ulcers. Following his late master, Maydl, he records, during the period from 1891 to 1904, 34 gastric or pyloric resections for benign disease, with 2 deaths, a mortality of a little less than 6 per cent. It is not his operative results that I mistrust, for they are brilliant and speak for themselves; but it is the conclusions which he deduces from a study of his cases that I think should be accepted with caution. He states that his microscopic studies (confirmed in most cases by Hlava) showed that of his patients whose stomachs were resected for gastric ulcer, supposedly benign, no less than 26 per cent. had commencing malignant degeneration. He points out, moreover, that of the 41 patients whose stomachs were resected, between 1891 and 1901, for carcinoma, not one is alive now; whereas, of the 4 patients whose stomachs were resected in the same time for supposedly benign disease, which was, however, afterward found by the microscope to be malignant, but in an early stage—that of these 4 patients, the first is well eight years after the operation, the second is well four years after the operation, the third is still too recent to count, and only 1 died of recurrence, and then only after two circular resections of the stomach. In all fairness I must add that Mayo Robson[39] has recently reported, among 97 patients operated on by gastroenterostomy for benign disease, no less than 4 who developed cancer at periods of from one to three and one-half years after the palliative operation; and that Czerny's clinic[40] furnished 3 such results among 83 operations for benign disease. But then it must not be forgotten that if these patients had been treated by the more dangerous method of gastrectomy, it might have been their fate to succumb to the operation—a result immeasurably worse than the remote and improbable chance of carcinomatous change several years subsequent to operation. So that while it is right to acknowledge the force of the arguments advanced by the advocates of gastrectomy as a prophylactic measure, I think it is a saner course to limit the more dangerous operation of resection to the more serious condition of acknowledged malignancy, and when there is doubt as to the existence of malignant disease to perform the more extensive operation, not as a routine measure, but only in carefully selected cases.

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[35] Berl. klin. Woch., 1882, xix, 39.

[36] Ibid., 1906, xliii, 1207.

[37] See cases reported by Bidwell, AMER. JOUR. MED. SCI., 1899, ii, 260; Demoulin et Tuffier, Bull. et mem. de la Soc. de chir. de Paris, 1899, xxv, 833; Robson and Moynihan, Diseases of the Stomach, New York, 1904, p. 386; Wallis, Brit. Med. Jour., 1906, ii, 73.

[38] Operativ. Behandl. d. Chron. Magengeschwürs, Prag., 1904.

There remains for discussion the choice of operation on patients who are undeniably affected with carcinoma. Until operation is more frequently undertaken in the very early stages of the malady, gastroenterostomy must still be the operation most often adopted. It has been rare in my experience, as I think also in that of other surgeons, for a patient in whom cancer has been certainly diagnosticated before operation to present conditions admitting of gastrectomy. The operations of gastrectomy that I have done myself have been on patients in whom the existence of malignant disease was merely suspected, but not certainly known, before the abdomen was opened.

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[39] First Congress of Internat. Soc. of Surgery.

[40] Petersen u. Machol, Beitr. z. klin. Chir., 1902, xxxiii, 297.

A palpable tumor felt before operation will naturally suggest cancer; but the case reports already referred to, in which such masses have been known to disappear after gastroenterostomy, prove that all palpable tumors are not carcinomatous in nature. I have urged elsewhere[41] that in this class of patients exploratory laparotomy finds its most legitimate field. The statistics from the clinics of Krönlein and Mikulicz, recently compared by Moynihan, show that patients who have undergone an exploratory laparotomy in which no further operative treatment was possible, actually lived longer than did those whose disease was so far advanced as to make even an exploration unjustifiable, or those who entirely refused an operation of any kind. In very many operations, moreover, which are commenced as explorations merely, it is found possible either to remove the growth or, at least, to perform a palliative operation which will materially prolong life and relieve suffering. Surgeons, I fear, sometimes lose sight of the fact that this and not the production of statistics is the end and object of surgery.

We have a choice of radical operations for cancer of the stomach, dependent upon the extent of the disease. Total gastrectomy, though it has been adopted in no less than 27 instances, must remain, I think, an operation of the utmost rarity. Personally I do not approve it. Though 17 patients survived the operation (a mortality of over 37 per cent.), I think it probable that the mortality will rather increase than decrease if the operation be indiscriminately employed. Subtotal gastrectomy, as it is called by the French, is in my opinion as extensive an operation as it is wise to adopt. I have successfully (?) removed as much as two-thirds of the stomach, always leaving the cardiac end and the "greater tuberosity" untouched. The operation of pylorectomy, or partial gastrectomy as it is now more accurately called, is that form of excision which is most generally applicable. The mortality of subtotal gastrectomy was 30 per cent. in 20 cases collected by Paterson—all he could find records of; while the average mortality of partial gastrectomy for carcinoma is, as we have already seen, from 26 to 28 per cent.

Adhesions, I think, a more formidable objection to radical removal than size of the tumor or lymphatic involvement. Especially is this true when the adhesions involve the pancreas. Enlarged glands should always be removed when possible, but as they are frequently the seat merely of inflammatory, not of malignant changes, their number need not discourage the surgeon from attempting radical cure by gastrectomy, provided the tumor is fairly movable. Thus Taylor[42] did a partial gastrectomy in a woman, aged sixty years, excising with the tumor *en masse* some of the right rectus abdominis muscle to which it was adherent, along with some enlarged glands in the gastrohepatic omentum. The patient recovered, and microscopic study of the tumor showed that it was of purely inflammatory character. Still more extensive operations have been performed when the growth was really carcinomatous. Moynihan[43] has twice (once with success) excised a tumor of the stomach with the corresponding portion of the transverse colon which was implicated in the malignant growth; and a similar operation has been successfully performed by Childe[44] (for unavoidable destruction of the blood supply, not for carcinomatous invasion), who also excised a layer of pancreatic tissue.

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[41] N. Y. State Jour. of Med., 1906, vi, 64.

[42] Indian Lancet, 1906, i, 668.

For the restoration of the gastrointestinal canal after gastrectomy I much prefer the usual posterior retrocolic gastroenterostomy to either of Billroth's methods, or to Kocher's operation of lateral implantation of the duodenum into the posterior gastric wall. In some patients, I think, it is wise to do this part of the operation first. Paterson also has recommended this course, and suggests that while the gastroenterostomy is being done, a piece of the suspected growth can be frozen and examined under the microscope. The plan suggested a decade or more ago by Tupolske, of doing a gastroenterostomy at one sitting and postponing the gastrectomy for several weeks, is open to the objection, as Mayo Robson[45] has pointed out, that the patient may be so much relieved by the palliative operation that he will decline further treatment. In one of Paterson's patients, however, treated thus by a preliminary gastroenterostomy, good health persisted after four years; so it is probable that in this individual case it was wise for the patient to decline excision of the supposedly malignant growth.

When the carcinoma has progressed very far, so that not even gastrojejunostomy can be done, the operation of jejunostomy will relieve suffering and may prolong life. I do not think the profession in this country appreciate the degree of alleviation of suffering which this procedure affords. The fistula should be established by a technique similar to that employed by Witzel in performing gastrostomy. In such patients, after opening the abdomen, no time should be lost in idly exploring the stomach and duodenum. We should know before commencing the operation what it is desired to accomplish, and the primary loop of the jejunum should be immediately sought and attached in the manner advised to the anterior abdominal wall. I prefer the incision splitting the fibers of the left rectus muscle. Feeding should be begun almost immediately, in moderate amounts, such as would be given to a starving man under other circumstances. Rectal alimentation may also be employed, but, except for the saline solution which can be thus absorbed, it is of little value.

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[43] Trans. Chir. Soc. London, 1906, xxxix, 84.

[44] Brit. Med. Jour., 1906, i, 194

[45] Ibid., 1900, i, 696.

In conclusion, I may say:

1. By timely operation for the various causes of persistent indigestion, many patients will be saved from developing gastric carcinoma.

2. Early diagnosis of gastric carcinoma being so extremely difficult, and radical removal being only highly promising when an early diagnosis has been made, partial gastrectomy should be an operation limited to cases of malignant disease operated on for symptoms of pyloric obstruction or other gastric affection supposedly benign, in which the existence of cancer, though perhaps suspected, cannot be certainly diagnosticated before operation.

3. In cases of gastric carcinoma moderately advanced gastroenterostomy should be preferred to partial gastrectomy.

4. When the indication is to prevent starvation, jejunostomy should be performed.

HYDROCEPHALUS COMPLICATING EPIDEMIC CEREBROSPINAL MENINGITIS.

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Hydrocephalus complicating or occurring with acute cerebrospinal fever is of the greatest practical and clinical importance. It has been observed since the disease has been recognized. In modern times a description of it begins with Foerster, in 1883, but a more extended consideration is given it by von Ziemssen and Hesse. These authors, in describing an epidemic of cerebrospinal meningitis, with autopsies, record cases of associated hydrocephalus in which the duration of the disease was comparatively short, from eight to nine days, and in which autopsy showed a dilatation of the ventricles with purulent content. Other cases with autopsies are recorded by these authors, in which the disease had a protracted course, from seventeen days in one case, to ten weeks in another, and in which autopsy revealed dilated ventricles of the brain filled with purulent content. Cases were observed in young children as well as in adults. A classical, clinical analysis of their cases showed that the hydrocephalus was accompanied by headache, vomiting, unconsciousness, and convulsions, and that this symptom-complex in some cases remitted, to return at irregular intervals.

Von Ziemssen[1] lays particular stress on the paroxysmal nature of the symptoms which accompany hydrocephalus complicating cerebrospinal fever. They consist of paroxysms of headache, pain in the neck, vomiting, unconsciousness, and convulsions. Hart[2] gives the clinical history and autopsies of cases of cerebrospinal meningitis in which he mentions the autopsy finding of dilatation of the ventricle. Otherwise he does not speak at length of the condition. Some of his cases, however, showed trismus and dilated pupils in the later stages of the disease. Heubner[3] takes up the subject of hydrocephalus occurring in cerebrospinal fever, but in a very terse and limited manner. Recent publications on cerebrospinal fever make very little note of this, it seems to me, most important condition. From my study of the condition I am convinced that our therapy must be guided to a certain extent by an early recognition of this most important state of the brain in this disease.

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[1] Handbook of Infectious Diseases, vol. ii.

There are three distinct conditions under which dilatation of the ventricle, or hydrocephalus of an acute nature, may occur in cerebrospinal meningitis of the epidemic type. I refer more especially to the meningococcic epidemic form.

Clinically the most important form of acute distention of the ventricles—we may call it acute temporary hydrocephalus—is that which occurs at the very onset of the disease. It does not occur in every case, but in the cases in which it does occur an early recognition of its presence, I am convinced, is the means of saving the life of the patient. I may illustrate more forcibly the point of my argument with a definite clinical case.

Case I.—A child, aged five years, was taken with a slight sore throat, a high febrile movement, and vomiting. The fever continued for twenty-four hours; there was some headache, but not to an extraordinary degree. He was sitting up in bed and playing with his toys; suddenly, without warning, he became pale, the pulse rapid and feeble, the temperature rose, after having been previously depressed, to 105°, the surface became cool, the child lay back in a collapsed condition, with shallow breathing, rapid, feeble pulse, becoming quickly unconscious, and in this condition was seen by the attending physician. I had the privilege of seeing this patient. I found a child of delicate nutrition, unconscious, with sighing, labored respiration, rapid, feeble pulse, surface cold and clammy, with a very few minute petechiæ on the inner sides of the thighs and arms, scarcely noticeable, pupils dilated; percussion of the skull on either side giving a hollow, tympanitic sound, which has been described by Macewen as evidence of distention of the ventricles by fluid. The pulse in this case was 150, the respiration sighing and irregular. The diagnosis of distention of the ventricles by fluid thirty hours after the onset of cerebrospinal fever was made.

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[2] St. Bartholomew's Hospital Reports, 1876.

[3] Eulenburg's Encyclopädie.

Lumbar puncture in this case confirmed two facts: First, that the case was really one of cerebrospinal fever of the meningococcic type; and, second, that the fluid in the subarachnoid space was under great pressure, fully 50 c.c. being withdrawn before the pressure seemed normal. The pressure was not measured, inasmuch as the manometer was not at hand. After the puncture the conditions of collapse, cardiac depression, and cerebral prostration improved; after days the mental condition cleared up and the patient made an uncomplicated and speedy recovery.

Another example of this form of acute distention of the ventricles threatening life, seen by me lately in conference with the attending physician, was the following:

Case II.—The patient was a male child, aged two years and three months. Thirty-six hours before the onset of the symptoms the patient was perfectly well, playing around the house. The child was taken with a high febrile movement, vomiting and headache, being intelligent enough to complain. It rapidly became more stupid and when seen by me was in the following condition: It lay completely prostrated, pale in the face, and the lips blue. The pulse was rapid, 130 odd, very feeble in one arm and at the other wrist not to be felt. The child could be roused, but rapidly sank again into a stupor. There was great hyperesthesia and slight rigidity of the neck, and petechiæ over the body. Percussion of the skull revealed a tympanitic note, as described by Macewen, on both sides, demonstrating a distention of the ventricles with fluid. Pupils dilated, no paralysis, no Kernig sign, no Babinski sign. Lumbar puncture made forty hours after the onset of the disease, revealed a fluid of milky turbidity, containing Diplococcus meningitidis, and evacuated under great pressure, continuing to flow to the extent of 40 c.c. before the pressure became normal. In this case, also, after puncture the pulse recovered tonicity, the sensorium became brighter, the child no longer exhibited attacks of collapse; once or twice after the puncture it vomited. The sensorium became progressively clearer until complete consciousness returned, within twenty-four hours.

In these cases the collapse, added to the distention of the ventricles, may be so great that, as in the first case, the paralysis seems to be almost complete. In very severe cases the typical symptoms met in the course of the meningitis, and which in these cases, reappear after the distention of the ventricles is relieved, may be absent. I refer to neck rigidity and Kernig's sign or opisthotonos. In the first case the patient was distinctly limp, and the collapse and prostration were so great as to resemble very closely what is seen in apoplexy and internal hemorrhage. In the second case, however, there was some rigidity and considerable reaction to irritation, a hyperesthesia which was absent in the first case.

Case III.—Female, aged five years. The child had measles when one year old, and whooping-cough when two years old. Otherwise the general health had been good. The present illness began suddenly on March 16, 1906, with severe frontal headache and vomiting. The vomiting was repeated several times, was induced by food and drink, and was not projectile in character. There was slight fever on the evening of the first day, but no chills or sweats. The mother noticed no twitchings, convulsions, or palsies; but did notice stiffness of the neck on the following morning. The child refused nourishment, and was constipated.

On admission to the hospital on March 17 (at the end of thirty hours) the following notes were made: The child is prostrated, cyanosed, stuporous, apathetic, hyperesthetic, and irritable at times. She moans continuously, and grinds her teeth. The knees are drawn up; the head is not retracted. The left pupil is larger than the right; both react to light. There is internal strabismus of the right eye; ptosis of the left lid. No hippus. Mydriasis on flexion of the neck. Slight injection of the conjunctiva, and slight discharge. There is left facial paralysis; and tenderness over the tragi. Slight rigidity of the neck. Bilateral Macewen sign. There is general enlargement of the lymph nodes, especially of the cervical. There is a rapidly appearing tache on the skin, and a few minute rose-colored spots on the abdomen and back, resembling insect bites. The breathing is rapid, shallow, and irregular. There are numerous coarse rales throughout the chest and showers of subcrepitant rales at the end of inspiration in the interscapular regions. The heart is normal. The patellar reflexes are not obtained. The abdominal reflexes are equal and active. Kernig sign with dorsal flexion on both sides at 180°. No clonus. There is involuntary discharge of the urine and feces. There is a leukocytosis of 26,000. Lumbar puncture was performed, 25 c.c. of turbid fluid being obtained. The pressure at the beginning was 40 cm., at the end 3 cm.

March 19. General condition is much improved since lumbar puncture. She has no headache and no pain, and is propped up in bed. Pupils are of medium size and react normally. There is still a purulent discharge from the right conjunctiva, and a slight internal strabismus of the right eye. Flexion of the neck is still tender and causes slight mydriasis. There is no longer ptosis of the left lid, but there is still slight weakness of left side of face. Macewen percussion note is still present. Numerous coarse rales are still present in both lungs. Breathing is almost regular. There is a delayed tache. Knee-jerks active. Babinski is present on both sides. There is no Kernig sign, but there is dorsal flexion of the great toe at 180°.

21*st* (sixth day). General condition is improved. Patient is intelligent as well as conscious. She says she feels no pain.

The head is held slightly retracted. The child continued to improve. Recovery was complete by April 15.

This form of acute distention of the ventricles, or acute hydrocephalus at the outset of the disease, does not occur in all cases of cerebrospinal fever, but when it does occur it is an important condition to recognize, for its continuance may mean the fatal issue of the case, or an unfavorable modification of its course. In this respect, it seems to me, modern clinical methods are enabled to point the way clearly to therapeutic interference—that is, the relief of the distention. In these cases there seems to be no permanent injury to the brain, if the patient recovers, as a result of the enormous pressure which has been exerted upon the ventricle walls. I may call this condition acute hydrocephalic distention of the ventricles of the brain by exudate in the initial stage of cerebrospinal fever.

The second form of hydrocephalus, which was at one time, and in some of the text-books on neurology today is, thought to be the peculiar and only form occurring in children, is that which occurs in the course of cerebrospinal fever, attacking infants below two years of age. In a paper which I published last year on posterior-basic meningitis I illustrated this form of hydrocephalus and remarked that it is the most frequent outcome of cerebrospinal fever in young infants and children, occurring in that form of meningitis of the cerebrospinal type which attacks the structures at the base of the brain in its posterior half. This form of hydrocephalus does not necessarily occur at the outset of the disease, but may supervene after the disease has lasted some time.

The symptomatology of this form of hydrocephalus is very difficult to separate at the outset from the symptom complex of meningitis proper. In infants attacked with meningitis the exudate in the ventricles seems to find room and the ventricles readily expand, especially if the child is below one year of age, on account of the condition of the skull, the sutures, and the fontanels. The result is that tension in the ventricles is not so apparent in early life as it is later, when the sutures and fontanels are closed.

Percussion of the skullcap in these cases does not give the tympanitic note referred to in the first set of cases, but rather gives the impression of a sac with fluctuating fluid contents.

Lumbar puncture relieves the pressure in these cases, as is indicated by the collapse of the bulging fontanel, but the fluid soon reaccumulates and symptoms, such as unconsciousness, convulsions rigidity, soon return. In other words, there is no restraint, it seems, upon the re-accumulation of fluid.

In children of a later period of life, from one and a half to two and a half years of age, the fontanels and the sutures being closed, the Macewen tympanitic note may be obtained in the course of the disease when the hydrocephalus occurs. And even in these cases it seems that the brain tissue is still so soft and resilient that reaccumulation of fluid easily occurs in a great number of cases. In a word, hydrocephalus occurring in the course of meningitis and not at the outset, in infants and children, is the natural sequence of the disease in a large majority of cases.

A third clinical form of internal hydrocephalus occurring in cerebrospinal fever is that which has already been referred to and which has been described by older writers. It may occur in any period of the disease after the first week. The patients usually are doing apparently well, they may have recovered from the severity of the onset of the disease, and may not, at any time, have been unconscious. There may be an intermittent febrile movement, the temperature at times being quite high, 104° or 105°, or even higher, falling within a few hours to the normal or subnormal, to rise again after a few hours to the original height. A patient running this intermittent or even irregular type of fever may be progressing favorably; that is, although complaining of headache, the child may be conscious, it does not vomit, and the headache, though severe, is mostly of the occipital type. Gradually the patient becomes stupid; there is vomiting and an increase of rigidity; the patient still may answer questions and answer them rationally. If, at this stage, percussion of the skull is carried out, as I have described, a slight tympanitic note may be heard over the ventricles, on one or both sides. This is the first indication that a distention of the ventricles with exudate is becoming extreme and that a hydrocephalus is forming. Relief at this stage of the symptoms may mean the saving of the patient, and overlooking the symptoms may do irreparable damage. Such a case is the following:

Case IV.—A boy, aged nine years, was taken with the typical symptoms of cerebrospinal fever of a moderately severe type. He had been perfectly well previously, had partaken of some indigestible food, complained of headache, and vomited once. He then developed rigidity and other symptoms of cerebrospinal fever, and began to run a temperature which ranged at the outset from 102° to 105° or 105.5°, with daily remissions to the normal or subnormal, a curve very closely resembling a typhoid fever in the third week. This boy, being highly intelligent, was conscious during the whole course of the disease up to a certain period. On the twentieth day of the affection, when he vomited for the first time, the headache, which had been quite severe and had been accompanied by pain in the abdomen of a very severe type, became still more insufferable. Chilly sensations were present. The intervals of consciousness and rationality became less frequent, he passed gradually into a state of sopor, having been quite conscious twelve hours previously.

Percussion of the skullcap revealed a tympanitic note on the left side, showing distention of the ventricles. Puncture in this case revealed a slightly turbid fluid under very great pressure.

falling to the normal only when 40 c.c. had been removed. After the relief of the distention of the ventricles the patient became quite bright, although talking constantly. He took nourishment, still complained of headache and pain in the ears, but was more rational. The improvement was steady, the pain and headache diminished day by day, the temperature fell to the normal, and the patient made an uncomplicated recovery.

It seems from a study of the temperature chart that apparently—and this patient was watched very closely—the correct moment was chosen to relieve pressure, for after the puncture the temperature rose to its highest, 105.5°, then fell to the subnormal, and rose to the normal and remained there until complete convalescence. Before the relief of the pressure the pulse was not only irregular but high; it became slower and more regular after relief of the hydrocephalus or acute distention.

The histories of cases illustrative of this type, in which recovery takes place, are given here simply to emphasize how early and closely these cases may be diagnosed, although it must be confessed that not in all cases is the outcome so fortunate. It is in these very cases of prolonged course that von Ziemssen has shown how hopeless is the outcome in a great many instances.

Of especial interest are the very mild cases of cerebrospinal meningitis in which the symptoms of the disease are so equivocal as to mislead even a careful observer. Such patients may make apparent complete recovery from an initial attack and after an interval of a month develop symptoms of meningitis of a severe type, with acute internal hydrocephalus superadded from the outset. Such a case was recently under my care, the history of which is as follows:

Case V.—Admitted with an initial attack of cerebrospinal meningitis of three weeks' duration. On admission there was no indication of distention of the ventricles. Active symptoms subsided two days after admission, temperature remained normal for ten days, when he was discharged cured apparently. Readmitted with a history of freedom from symptoms for forty days, when there was a recrudescence of symptoms of cerebrospinal meningitis, with signs of mild and then increasing hydrocephalus. In spite of repeated puncture there was no relief of symptoms, and death resulted from inanition.

Case VI.—A male, aged seven and a half years, was admitted with signs of distention of the lateral ventricles on the fourth day of the disease. The symptoms of acute hydrocephalus were relieved. There was a return of the symptoms of hydrocephalus on the twenty-third day of the disease, as evinced by exacerbation of headache, dilated pupils, sopor, vacant expression to face, hyperesthesia, and vomiting, with Macewen percussion note over the ventricles of the brain. Remission occurred and then exacerbation of symptoms on up to the fortieth day of the disease. Death occurred on the forty-first day. Temperature was normal from the thirtieth day of the disease, at times subnormal. The postmortem revealed chronic hydrocephalus, marked dilatation of the lateral ventricles, and marked distention of the iter.

In this case postmortem there evidently was no obstruction to the escape of fluid from the ventricles; this could not be a cause of the hydrocephalus.

Case VII.—Female, aged three years seven months, admitted March 8, 1906. The child was always strong and healthy. The present illness was of twenty-four days' duration. The onset was sudden, commencing with headache; later vomiting. Fever from the onset. Headache lasted for a few hours; followed by deep stupor. Remained stuporous for four days. Then lumbar puncture was performed; following this she regained consciousness. Mind was clear until two days ago, when stupor developed again, and condition has not changed since. Vomited the first day. Father does not know if it ever was projectile in character. Vomited usually after food or medicine. Has not vomited since the first day. Fever was very high, 105°, first day; very irregular in type. Temperature more moderate lately than it has been. No history of any chill, sweats, or convulsions. No grinding of teeth. Child lies with mouth open all the time. No twitchings or convulsions. General muscular rigidity, particularly at the neck and knees since onset. Has not been delirious since day of onset. Very irritable and hyperesthetic all the time. Hearing somewhat impaired for the last two days. Takes nourishment well. Sleeps fairly well at night, occasionally requires bromides. No history of night shrieks. Bowels at first constipated. For two weeks until two days ago, five to six watery movements daily. No mucus or blood (one drop of blood on one occasion). Odor bad. Was getting potassium iodide and bromide. Medication then stopped and bowels improved. No vomiting at the time. Had a rash on the body at this time, large patches, general urticaria. No eruption at the onset of the disease. Urination: increased frequency and amount. Has had loss of control of the rectal and vesical sphincters. During the first week there was weakness of left arm and left leg for a few days. Not since. Patient has had periods of improvement during present illness. Child sat up in bed and sang and played, although some temperature persisted. Condition much worse for past two days. Child hears and sees. Lumbar puncture was performed three weeks ago. 30 c.c. of turbid fluid was obtained.

Chief complaints: Restlessness, irritability, fever, rigidity of the neck, and flexion of the knees.

Physical Examination. General condition, fair. Patient is fairly well nourished, looks sick. Lies on right side, with knees drawn up. Is stuporous, irritable when touched, and vicious.

The neck is rigid. No mydriasis on flexion. Marked Macewen sign, bilateral. The pupils are equal, moderately dilated. Right and left internal strabismus. Conjunctivæ negative. Left facial paresis. The skin is hot and sensitive to touch. There is a tache. The heart and the lungs are normal. The knee-jerks are not obtained. Bilateral Babinski sign. Kernig sign, with dorsal flexion, on both sides, 180°. No clonus.

March 9. Both ear-drums slightly reddened. Leukocytosis of 20,000. Lumbar puncture performed on account of restlessness and presence of Macewen sign. Puncture was made in fourth space. 30 c.c. of slightly turbid fluid obtained. At beginning pressure was 25 mm.; at end pressure was 3 mm. Depth of introduction 3 cm.

10*th*. Restlessness the same. Macewen still present.

14*th*. Blood. Leukocytes, 18,000. Hemoglobin, 78 per cent.

15*th*. Child is irritable. There is internal strabismus of left eye. There are occasional involuntary discharges of urine and feces. Child looks worse. Neck is slightly retracted, fever continues. There is a Macewen sign present. Neck is rigid. There is mydriasis on flexion of neck. Breathing is irregular and cerebral in type. Pulse is irregular. There is a Kernig sign on right side and on left side with dorsal flexion of 165°. There is a bilateral Babinski sign.

16*th*. Lumbar puncture. Needle introduced 2.9 cm. Pressure (initial) was 22 mm.; final pressure oscillated between 7.5 and 15 mm. About 20 c.c. of a slightly turbid fluid was withdrawn.

April 16. Child has steadily improved, sits up, sings and plays with children, though she has signs of a persistent hydrocephalus. Her nutrition also has improved.

These are cases to which the older writers referred as being of discouraging prognosis. The symptoms of hydrocephalus are progressive under every treatment, and are accompanied by extreme emaciation, clouding of the intellect, symptoms of encephalitis, such as paresis of the facial muscles and extremities, volitional tremors, vomiting, and convulsions.

One of the principal symptoms which is striking in subacute meningitis in which hydrocephalus supervenes is the progressive emaciation, in spite of nourishment which is sufficient to maintain nutrition. These patients will take large amounts of nourishment and will emaciate progressively in spite of the food elements which are retained in the body. Emaciation in these cases differs from the emaciation which is seen in the course of any acute disease, or in the course of favorable cases of cerebrospinal meningitis, in that it is extreme and seems to me to be of a trophic nature.

Toward the latter end of these cases of hydrocephalus the patients develop symptoms of encephalitis, such as paresis of the ocular and facial muscles and those of the extremities, and, what is especially noteworthy, volitional tremors of the muscles of the face, jaw, and extremities.

From what has been said there has been an indication that with the symptoms of meningitis a condition of hydrocephalus yields a distinct symptom complex. Given a case of meningitis in the course of which, after the initial stage has passed, the patient is improving gradually as to his sensorium, the temperature is falling, as in one of the cases related, there is a dulling of the intellect, vomiting, dilatation of the pupils, strabismus, a vacant stare to the expression, sometimes even in older children, a depression of the eyes, showing the sclera—in such cases the hydrocephalus seems to have been well inaugurated.

The early symptoms are those indicated by percussion of the skull. A patient who is apparently doing well, who may have had a normal temperature, will develop an ephemeral rise of temperature, sopor, headache, and vomiting, and percussion of the skullcap, very early in the disease, will reveal a tympanitic note, showing not only dilatation of the ventricle but an accumulation of fluid within. This I consider one of the most useful guides in the subacute cases of meningitis in which there is threatened or well-established hydrocephalus. Today we need not wait until even the tyro can diagnose an accumulation of fluid in the ventricle in acute cases, but by the aid of percussion of the skull and the study of the symptoms we can diagnose the condition quite early.

A symptom of moment is vomiting. Vomiting in the course of meningitis of the subacute type is a very ominous symptom, especially if repeated and if it occurs independently of the ingestion of food, that is, at the moment after the food is taken into the stomach.

Dilatation of the pupils and strabismus, especially in younger children, are other symptoms. The pulse in all cases of hydrocephalus is irregular, both in rhythm and force. It was formerly thought that hydrocephalus could occur only with a normal or subnormal temperature, but a study of temperature curves shows that hydrocephalus may occur with an intermittent high temperature, which intermits to the subnormal daily; or it may supervene in a patient whose temperature is normal; or when once inaugurated, the temperature may run an intermittent subnormal course.

Of especial interest in relation to early dilatation or acute distention of the ventricles is the effect of extreme pressure on the pulse and respiration. In those cases in which the hydrocephalus supervenes at the onset of the disease the patient goes into a condition of collapse, the pulse may be rapid and thready, the surface is cold and ashy in hue, the pupils are dilated, respiration is sighing in character, the patient exhibiting all the symptoms of cardiac collapse. It is wonderful to what extent the ventricles of the brain may be distended and still the patient rally from the symptoms, showing in the recovery no signs, so far as percussion of the skull or the mental state are concerned, of the previous great, and what must have been life-threatening, pressure.

Of interest also are those cases in which, in the course of the disease, when the symptoms are improving, there is a development of all the symptoms of hydrocephalus of the mildest type, as shown by percussion of the skull, the intervening sopor, dulness of intellect, irregularity of pulse, and the rapid recovery of the patient after interference. I have no explanation to offer as to why these patients do not continue to develop the symptoms of fully developed hydrocephalus, and why an exudate which suddenly threatens to increase should cease at a certain point and leave no marks or ill-effects of the distention.

METHODS OF DIAGNOSIS. I have referred quite frequently to percussion of the skullcap and its utility as a means of early diagnosis of hydrocephalus supervening either at the outset or in the course of meningitis. I have followed the method of percussion through two epidemics of cerebrospinal meningitis and I am convinced that its more general adoption will lead to better results in the early diagnosis of this symptom complex than has been hitherto possible. Macewen was the first to point out, in conditions other than meningitis, the utility of percussing the skullcap in the diagnosis of hydrocephalic effusion into the ventricles of the brain. Children at the age at which the ventricles and sutures are not yet closed, as already referred to, do not lend themselves to this method of diagnosis of hydrocephalus, but in children and adults in whom the sutures are ossified and the skull fully formed, utilization of the Macewen percussion of the skull in the course of meningitis will enable one to detect at a very early stage a hydrocephalic effusion into the ventricles. In some cases, as in some of the histories subjoined, we may be able to detect that the effusion is unilateral at first and then may become bilateral. At times percussion of the skullcap will reveal effusion in the ventricle, and after an interval of a few days this percussion note may not be so indicative of effusion—that is, it may not be so tympanitic, then may return to the tympanitic note after a day or more. The cause of this I am unable to explain. It is simply a clinical observation.

A word may be said here as to the cause of hydrocephalus in cases of cerebrospinal meningitis. Joslin, in an article on internal hydrocephalus complicating cerebrospinal meningitis, gives the autopsy findings in one case of his own and in a number of others, in which there was an occlusion of the foramen of Magendie or Mierjewski, or a matting together of the structures at the base of the brain, thus preventing the escape of the fluid, either transudate or exudate, from the ventricles through the foramen of Monro and then of Magendie to the subarachnoid space. In a paper on posterior-basic meningitis which I published in 1905, I related one case of posterior-basic meningitis in which this foramen was also closed. Whereas, in some cases of hydrocephalus the mechanical closure by exudate of this foramen may explain the retention of fluid in the ventricles, it certainly does not explain why hydrocephalus occurs in these cases, for in the vast majority of cases of hydrocephalus which I have observed a lumbar puncture by a skilled hand could withdraw large amounts of fluid from the subarachnoid space, which argues against the existence of any obstruction of the foramina mentioned. In fact, I am inclined to believe that the closure of these foramina is an exceptional and accidental finding in a few cases, and that in most cases of hydrocephalus the cause of the hydrocephalus lies in the inflammatory changes in the bloodvessels and lining structures of the ventricles of the brain, for in no other way could we explain the acute cases at the very onset of the disease, and the subacute and chronic cases both in young and older children and adults, in which it is an easy matter to remove large quantities of fluid from the subarachnoid space and ventricles by lumbar puncture.

In those cases which have run a subacute or chronic course and in which internal hydrocephalus exists and the foramen of Magendie is either narrowed or closed, lumbar puncture will succeed in evacuating 10 or 15 c.c. of clear fluid under little or no pressure. In other words, the fluid flows drop by drop and ceases to flow after a time, uninfluenced by the cries of the patient or by respiration. In other cases in which the canal of Magendie is closed the puncture will evacuate but a few drops of clear fluid. In such cases it is not difficult to see that the hydrocephalus must not only persist, but its disappearance becomes impossible.

THERAPY. It is but proper to consider here, if only in a few words, the question as to whether we have succeeded in curing directly or indirectly by therapy any of the forms of hydrocephalus mentioned in this paper. In considering the question of therapy I have in mind only the procedure of lumbar puncture. In those cases which have been described in this paper in which hydrocephalus supervenes at the outset of cerebrospinal meningitis, lumbar puncture has seemed to me to be a procedure of great utility, not only in relieving the pressure and therefore preventing irreparable damage to so delicate a structure as the brain, but, in very severe cases, by relieving the pressure we relieve a set of collapse symptoms which, if allowed to continue, certainly are not devoid of danger to life.

In those cases in which an early diagnosis is able to detect threatened hydrocephalus in the course of the affection, in the second or third week of the disease, a single lumbar puncture has, in some instances, been sufficient not only to relieve the symptoms which were evidently caused by increasing fluid in the ventricles, but to arrest the progress of the disease at a critical moment by the removal of the excess of toxins or bacteria from the interior of the cranial cavity.

It is, of course, difficult to say in those cases in which a marked improvement has followed a single lumbar puncture, how much influence this one procedure had in arresting the disease. I have given a demonstration in this paper of some such cases, and I leave it for future investigation to prove whether there is any proof in this supposition. If the hydrocephalus, after a primary puncture becomes progressive, as in subacute cases, and there is a continued rise and fall of temperature, or even a normal temperature and continuance of vomiting, with headache, Macewen's sign—showing re-accumulation of fluid in the ventricles—dilalation of the pupils, extreme emaciation, and in time signs of encephalitis, such as volitional tremors and paresis, it has seemed to me that lumbar puncture has been of very little avail in a curative sense. What lumbar puncture fails in doing in these cases it possibly succeeds brilliantly in doing in other cases.

It is but proper to say that the studies which have given origin to this paper seem to me to have definitely fixed the indication for the procedure of lumbar puncture in cerebrospinal meningitis of the acute type. The indications hitherto for the performance of this operation have been exceedingly vague. Lenhartz[4] speaks of repeated lumbar puncture as a routine procedure in cerebrospinal meningitis. Netter, in the early history of the disease, was one of the first to insist on the utility of applying this routine procedure. I even went so far as to publish several cases in which I thought I had cured a series of cases by repeated lumbar puncture. More mature study and experience have taught me that the indiscriminate application of lumbar puncture to the treatment of acute cerebrospinal meningitis is of as little utility in itself as a curative measure and as productive of harm as would be the indiscriminate use of any therapeutic procedure. Lumbar puncture should not be performed with a view to evacuating purulent or semipurulent contents from the cerebrospinal spaces, for in most cases this of itself does not cure the case. It should also not be performed for severe headache, fever, or unconsciousness, and, it seems to me, only in fixed cases should it be performed for diagnostic purposes.

It should not be difficult to make the diagnosis of cerebrospinal meningitis of the acute type without resorting to lumbar puncture. Today lumbar puncture is not resorted to by me except on a definite indication which crystallizes itself into the principle that the chief element of mischief in cerebrospinal meningitis is the accumulation of fluid in the ventricles of the brain, their distention by this fluid, and the evil effects of the pressure of the infected fluid on such a large surface of so delicate a structure as the brain substance. I have seen numbers of patients with meningitis recover without lumbar puncture being resorted to. These patients were simply nursed, fed, and given drugs according to the indication of the case; they recovered without a sign or symptom of the evil effects of the meningitis, unless it were in certain cases in which at the outset of the disease deafness or an iridochoroiditis developed, fatal to the ear or eye. In the recent epidemics in New York the recovery of patients who have been treated without lumbar puncture is tolerably complete and satisfactory.

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[4] Münch. med. Woch., 1905.

I resort to lumbar puncture only at the outset of the disease, or in its course, when I am convinced that there is an accumulation of fluid in the ventricles; unless the signs which I have detailed in this paper are present, irrespective of headache, delirium, or high temperature, I do not puncture. I feel that such cases are not benefited by lumbar puncture, although I have punctured many simply to convince myself that everything available in human knowledge had been offered to the patient.

CARDIOVASCULAR REGULATION DURING AND AFTER OPERATION.

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A series of observations extending over four years in abnormal cardiovascular cases, with special view to the added light that accurate estimations of blood pressure throw on the results of previous methods of examination, has apparently developed some important medical questions, detailed and published elsewhere. But, in addition, another part of the work, perhaps more than half, being occupied with surgical cases, has seemed to bring out even more important possibilities. These observations were conducted in the general surgical, gynecological, and obstetrical wards of the Johns Hopkins Hospital, Baltimore, and in the Memorial Hospital, Richmond; they embrace observations before and during operation and throughout convalescence, varying through a variety of cases, those presenting some unusual or serious feature engaging special attention. I wish particularly to express my gratitude to Dr. George Ben Johnston, of Richmond, Va., for the privilege of observing his cases.

Obviously the preservation and proper regulation of the cardiovascular system is of pre-eminent importance in the successful conduct of any surgical case of more than the most trivial nature, and most fortunately it now lends itself to the closest scrutiny of any system and presents the fullest possibilities of accurate distinctions.

An estimation of renal capability must be of the vaguest nature, and postoperative suppression gives no good warning; nor can we foresee the lack of intestinal tone which may determine a postoperative ileus, or the presence of such gastric irritability which may induce fatal vomiting. The cardiovascular system, the most important, is fortunately the most accessible. An educated touch for arterial changes, percussion skill to outline cardiac dulness, the pulse rate and quality, but above all the accurate estimation of pulse strength, furnish data which, when properly correlated and interpreted, by correct premises and the experience of repeated observation, have a value which should hardly be put of second importance to a correct diagnosis of the local surgical condition. Of little value to the patient, however scientifically satisfactory, is a correct differential diagnosis between myoma and carcinoma of the fundus of the uterus, if the cardiac strength or vascular strain has been mistaken or slighted, and the patient dies of acute cardiac dilatation on the table or during the first few days after the operation.

The deductions herewith presented are not built on the occasional observation which, as in those hospitals where blood-pressure records are taken at all, may be made every twenty-four hours, or before and after operation; in these cases the observations were made at frequent intervals and continuously, and always by the same person, or my friend Dr. Briggs, who worked with me at the Johns Hopkins Hospital. As we were accustomed to observe cases alternately and found our observations constantly agreeing, the error of personal equation was minimal. Usually during operation the pressure determination would be made every two and a half minutes and continued as frequently, sometimes forty-eight hours after the operation. In some cases when rapid variation was noted, one-half minute observations were recorded. The observations were always charted, as originally recommended by Cushing, so that the variations were most graphic. The blood-pressure observations were always correlated with the other vascular and general symptoms, particularly the area of cardiac dulness, quality and rate of the pulse, character of the arterial wall, facial aspect, and subjective symptoms. In the earlier cases the narrow (5 cm.) band originally made with my instrument was used; in the latter cases the wide arm-piece was used. The narrow arm-piece gave too high absolute readings, but the degrees of variation were quite comparable. I continue to use my modified Riva Rocci apparatus made in portable form for greater convenience in general practice. The same instrument in continuous use for three years remains perfectly satisfactory.

The cases showing abnormal blood pressure naturally divide themselves into two main groups: (1) Those with an increase in tension, and (2) those with a low tension. Besides these there is a third miscellaneous group of abnormal cardiovascular cases with normal blood pressure, representing in general the moderate grades of failing compensation. For instance, a well-compensated valvular insufficiency or arteriosclerosis may show a systolic pressure of 180 mm. Hg., but as the heart begins to fail the tension falls and may be found normal at some point along the progressive downward course. This condition necessarily must be recognized by signs other than the absolute blood pressure, and should be suspected when, without a murmur or hypertension, the heart outline is enlarged, showing weakening and stretching of the muscle, or the general condition and appearance indicate failing strength, especially if the arterial wall shows thickening and bending, such as might have been produced by a preëxisting chronic hypertension, or if the pulse is irregular in force or rhythm or accelerated. Such cases demand especial care to minimize the strain on an already failing heart; yet, as they usually show no murmur and often no irregularity of pulse, and have a clear urine (though albumin might have been found some time previously during the initial period of hypertension), they go to operation unguardedly.

In general, the tendencies of the high and the low-tension cases correspond to the similar cases in medical conditions. The high-tension cases stand the added strain of ether anesthesia and operative manipulation poorly and tend to suffer cardiac dilatation, excessive hemorrhage, vessel rupture, or renal affections, while if they survive the immediate effects of the operation the convalescence is ordinarily favorable. When, however, high tension persists after operation, it should be watched closely for some weeks, and treatment should be instituted to lessen the strain on the heart and blood-vessels. This would give us the necessary clue to those unfortunate patients who apparently recover promptly from operations only to suffer from acute cardiac dilatation and sudden death or cerebral rupture from a few days to several months afterward.

The low-tension cases conversely, in general, stand the operation well if chloroform be not used, if hemorrhage be not excessive, nor peripheral irritation extreme; yet, as has also been found from a medical point of view by the incidence of tuberculous tendency in low-tension individuals, these subjects show a lowered resistance to infectious disease and are subject to pneumonia or local infection.

The general rationale of prophylactic and corrective treatment in these cases follows naturally, after their recognition, from an application of such measures as tend to counteract the existing condition and the avoidance of those procedures that would exaggerate it. Thus, when abnormal hypertension exists prior to operation, a preliminary reduction of the tension should be accomplished, which reduction should be maintained at least through the first part of the operation and throughout when hemorrhage cannot be easily controlled, as the tendency to hemorrhage is, of course (coagulation time being constant), in direct proportion to blood pressure. This reduction in tension can be easily and equably accomplished, in the majority of cases, by the exhibition of sodium nitrite, the vasodilator which has proved preferable in my experience.

Sodium nitrite is usually dispensed only as crystals or in solution. Three to five drops of a saturated solution is usually a satisfactory initial dose. Tablets of $\frac{1}{2}$ grain and 1 grain have proved more satisfactory than the solution; they seem very stable, as I am now using some a year old which have unimpaired effect in lowering tension. They may be used hypodermically or by the mouth.

Throughout operation these patients need careful watching; the Trendelenburg positions should be assumed only when absolutely necessary, and then moderately, the hypertensive tendency of asphyxia being avoided as much as possible by a free supply of air or even oxygen with the anesthetic, and care taken not to embarrass further the overtaxed heart in an abdominal operation by stuffing more gauze than is absolutely necessary under the diaphragm. If the pulse, which is usually slow in these cases, becomes much accelerated without a corresponding increase in tension, an attempt should be made, if possible, to see if the heart is dilating.

I have records of 10 cases of acute dilatation of the heart in hypertension cases, in 5 of which death resulted on the table or within the next forty-eight hours. In 1 of the 3 patients that lived the heart at a tension of 180 mm. dilated in three-quarters of an hour, the pulse jumped to 160, and the operation for carcinoma of the cervix was abandoned. This woman was hurried from the table with a clinical diagnosis of shock, yet the pulse tension was still 180 and the heart waving a wide impulse outside the nipple line. Although the woman ultimately died of the growth, the vasomotor condition at operation precluded the possibility of cure. Nitrite would have probably corrected this sufficiently to have permitted completion of the operation. The hemorrhage also in this case was almost uncontrollable and would certainly have been bettered by a reduction in tension. The other cases were similar, though varying in details.

A patient with arteriosclerosis and an anteoperative blood pressure of 190 mm. Hg. left the table, after a Gasserian operation had been abandoned on account of uncontrollable hemorrhage, with a hemiplegia on the same side as the opening in the skull, showing it was not from direct injury.

In these hypertension cases chloroform is not indicated, as has often been urged for arteriosclerosis, for, although chloroform does lower blood pressure, the same effect can be produced by the vasodilators without the marked inhibitory and toxic action of chloroform. The convalescence, as mentioned above, is usually uneventful unless a rapid return of very high tension induces cardiac dilatation or secondary hemorrhage, the latter being especially dangerous if internal, of which consecutive blood-pressure observations afford the best indication.

Low-tension cases in general stand operation well, with proper precautions for the avoidance of shock and undue hemorrhage, and to the surprise of the anesthetist often leave the table with an improved pulse. This, in view of the hypertensive tendencies of operation and anesthesia, as just reviewed, is easily understood. Their low resistance, either dependent on the low tension or merely associated with it, may and should be corrected after operation by active stimulation to prevent the susceptibility to infection.

These low-tension cases seem to lack resistance to infection, and pneumonia, local abscess, thrombi, etc., seem to occur more frequently among them. Strychnine in repeated tonic doses, about $\frac{1}{40}$ grain for adults, is apparently most beneficial. The preliminary purging and fasting should be less stringent and nourishing food should be given as soon after operation as the gastric condition will permit.

That blood-pressure observations afford the earliest and surest indications of oncoming shock or excessive hemorrhage during operation can, I think, be no longer disputed. As consecutive blood-pressure observations during operations in serious or lengthy cases come into more general use, fewer cases of profound shock will develop under surgical manipulation. Pulse rate alone gives tardy and uncertain warning, and lessening tendency to hemorrhage, shallow respiration, and pallor are even more uncertain guides. A blood-pressure chart gives an accurate and graphic picture of vascular tone, and its value cannot be too greatly emphasized.

The few hours immediately following operation are fraught with many possibilities of dangerous turns and are often the most anxious ones. A study of the postoperative complications, in which the cardiovascular system plays the most important part, with the additional light of accurate knowledge of consecutive changes in pulse tension, affords a basis for classifying them into divisions which may materially aid their individual recognition. Usually the single symptom that is depended on to give warning or knowledge of a change in cardiovascular conditions is pulse rate, and a rise in rate or a rapid pulse is taken roughly to gauge the danger. Therefore (as detailed elsewhere, but yet unpublished) the divisions of tachycardia comprehend the majority of postoperative cardiovascular complications, and with the detailed association of other signs and symptoms may be briefly indicated as follows:

Hemorrhage. If hemorrhage is acute, the pulse rate does not usually rise to any marked degree until the loss of blood is excessive. It is not unusual to find a severe hemorrhage which has lowered the tension 75 per cent. in an hour, without raising the rate above 80. In slow hemorrhage extending over some days, as in some ruptured extrauterine pregnancies, the rapid pulse seems not so much an indication of loss of blood as of cardiac weakness from the impoverished, diluted blood. In a slow hemorrhage the hemoglobin percentage and pulse rate are better indications than tension, while in rapid hemorrhage the falling tension is not only the first but often the only indication until the degree is extreme. The character of the pulse is often full and bounding, and the height of the wave which is increased by the lowered tension may readily give rise to a mistake in palpation judgment, the large impulse giving the impression of strength. The small, thready pulse, due to a compensatory effort of the vasomotor centre to raise the tension by peripheral constriction, is of later development, and the loss in blood volume may escape palpation judgment. Pallor and subnormal temperature are the rule, and later the characteristic subjective symptom of dizziness. The area of cardiac dulness is not increased.

Shock. The onset of shock may not occur until after the patient leaves the table, perhaps through continued chemical or mechanical irritation of the severed nerves, and may then be severe. As already mentioned for the period during operation, blood-pressure determinations afford the earliest and most valuable indications of this loss of vascular tone. The temperature is subnormal, and subjective symptoms, such as great restlessness and anxiety, are common. Severe shock may exist without rise in the pulse rate.

Infection. Bacterial infection usually causes a rise in pulse rate which in some cases may be alarming, and when the possibility exists may cause suspicion of one of the two conditions just mentioned, shock or hemorrhage. But the tension does not fall and may even rise in response to the increased rate. This is true of pulmonary infections excepting tuberculous and local pyogenic infection at the site of the wound. I have repeatedly seen abdominal cases in which hemorrhage or shock was feared from the rise in pulse-rate; yet the continued observation of normal or increased blood pressure excluded both. The rise in temperature corresponding to the rise in rate and without a fall in tension confirms the diagnosis of infection. Of course, the later septic form of temperature which sometimes accompanies anemia following hemorrhage must not be taken to mean infection.

Cardiac Dilatation. This condition is more infrequently recognized, I believe, than any other postoperative cardiovascular complication; yet it is by no means rare and is of serious import, and, further, when recognized, lends itself readily to corrective treatment. From the number of cases I have personally seen, ten in all, I believe many of these cases, all of which show tachycardia, are treated with a view to raising the tension with the idea that they are in shock, or suffering from excessive hemorrhage, or that the failing vascular tone needs bracing. As a matter of fact, the rapid pulse often occurring in stout individuals in whom accurate palpation judgments are difficult, is mistaken for a weak pulse. Vasodilators are indicated, or *in extremis* even bleeding, and not the measures frequently used to increase the tension, such as the Trendelenburg positions, abdominal compression, adrenalin, caffeine, and saline transfusion. The registered pressure may be above 180 mm. Hg. Examination of the heart will reveal an increased area of dulness, especially to the left. The impulse, if visible or palpable, will be at or outside the nipple line, usually wide in area and wavy.

This paper is presented with a view of emphasizing the importance of giving more attention to the general condition of patients previous to operation and while the strain and shock of operative manipulation lasts, and to the value, during these periods, of blood-pressure observations, especially when controlled by other signs and symptoms. The recent advances in surgical methods and technique leave little to be desired along strictly operative lines, but still many patients go to operation with the old routine of a cursory auscultation for cardiac murmurs and urine analysis for albumin and sugar. During the operation, although excessive details of asepsis and technical surgical skill are observed, the general condition of the patient is merely followed as well as may be by the interne in what intervals he may find to feel hurriedly the pulse between filling his ether cone, swabbing out the mouth, and perhaps holding up a stubborn jaw. Yet the one-half to four hours of operation is the most severe and critical period in the lifetime of the patient, and likely to produce such variation above or below the normal pulse tension, as, taken in relation to the arterial and heart condition, may, on the one hand, determine arterial rupture or cardiac dilatation, or, on the other, respiratory failure in shock or hemorrhage.

After the operation a knowledge of these relations is of extreme importance in distinguishing and in guiding the treatment of certain postoperative complications, all of which may have the common and usually the only noted symptom of tachycardia; or when an anomalous slow pulse may give very deceptive indications when pulse-rate alone is studied. During that period of marked disturbance in cardiovascular routine it would appear that a more intimate medical knowledge, especially training in the observance of these surgical crises, would add much valuable light to the general aspect of the case, not interfering with but aiding and supplementing the strictly surgical procedure, and that every serious surgical case would be better off if given the benefit of accurate medical interpretation of cardiovascular indications. Such a course would have more far-reaching benefits than those immediately accruing to the individual patient, as it would tend more closely to cement and emphasize the natural relation which exists between medicine and surgery, and which finds truer expression in more extended co-operation.

CLINICAL STUDIES OF THE CEREBROSPINAL FLUID, WITH ESPECIAL REFERENCE TO PRESSURE, PROTEIN-CONTENT, AND THE NUMBER AND CHARACTER OF THE CELLS.

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More than fifteen years have passed since Quincke first performed lumbar puncture, yet the full scope of the measure for diagnosis remains undefined. The bacteriological results are satisfying when positive; a frankly purulent fluid, or one that shows grossly an increase in cell content, is almost equally so; but when the fluid flows water-clear, without an evident spurting to suggest increased tension, when it is bacteriologically negative and without marked cellular increase, the conclusion usually reached is that matters in the subarachnoid space are as they should be.

Quincke[1] gave us lumbar puncture as a clinical method; Krönig defined the pressure under which the fluid is held normally, and many later writers have pointed out conditions under which this is increased; Guillain and Parant[2] identified the protein constituents and showed their variation in disease; and Widal, Sicard, and Ravaut[3] are responsible for cell formulæ which they have held as indicative of special meningeal conditions. Of late, Donath,[4] Mott,[5] and Halliburton[6] have laid stress on the presence of cholin in the fluid during the course of some degenerative nervous diseases, notably paresis. Apart from these real aids are a number less well established: chromodiagnosis, cryoscopy, electrochemical resistance, the permeability of the meninges for certain drugs, the toxicity of the fluid, and its glucose content.

At present, consistent methods of work with each specimen seem much more needed than the discovery of new attributes. Clinically, apart from the gross appearance of the few cubic centimeters drawn off, there are four determinations of special value: (1) The pressure in the subarachnoid space, (2) the protein content, (3) the cell content, and (4) the cell character of the fluid.

Toison and Lenoble stated, in 1891, that the normal cerebrospinal fluid holds a very few lymphocytes, but many later workers—Mott, Schlesinger,[7] Widal, Sicard, and Ravaut—have considered it to be free from cells, and today the point is still undecided. In 1901,

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[1] Berl. klin. Woch., 1891, xxviii, 929.

[2] Rev. neurol., Paris, 1903, xi, 406.

[3] Ibid., Paris, 1903, 438.

[4] Ztschr. f. physiol. Chem., Strassb., xxxix.

[5] Brit. Med. Jour., London, 1904, i, 1554.

[6] Chemical Side of Nervous Activity, John Bale Sons and Danielssohn, London, 1901.

[7] Deut. med. Woch., 1904, xxx, 1022.

Widal, Sicard, and Ravaut noted a marked cell content in tuberculous meningitis; a little later Brissaud and Bernandet found numerous lymphocytes in tabes, Joffroy in paresis, and Sicard and Monod in syphilis of the central nervous system.

The method employed by these observers, namely, to judge of the presence and number of cells by examination of the sediment obtained on centrifugalization of the fluid, and determination of the cell number per oil-immersion field, has allowed such a large personal error that the satisfactory interpretation of statistics founded on it is impossible. Yet with but few exceptions the later workers have used it.

Laignal-Lavastine,[8] and a little later Joffroy and Mercier,[9] attempted exact quantitative counts, but the technique of the first is tedious, and we have no description of that of the others. In 1904, Fuchs and Rosenthal[10] presented a method whereby, with a weak aqueous solution of methyl violet and acetic acid, and a modified Zeiss counting chamber, a direct quantitative and differential count of the cells could, they asserted, be made. Kramer,[11] with this technique, and Simpson,[12] following nearly that of Laignal-Lavastine, have recently studied the spinal fluids of certain insane patients, most of them paretics.

The solution of Fuchs and Rosenthal greatly injures or destroys such red corpuscles as may be present, and, for reasons to be cited, these seem to be important in the count. I have, therefore, devised another method for the cases here reported: A saturated aqueous solution of methyl violet (5 B.) is drawn up in the tube of a 1 to 100 *melangeur* until it has filled four of the decimal divisions on the tube, and then it is followed with the fresh cerebrospinal fluid until the bulb of the instrument is full. This furnishes a mixture, slightly darker than ordinary "Toison solution," containing 996 parts in 1000 of the fluid for examination. In this way the leukocytes are stained violet, the erythrocytes preserve their hue, and with the usual blood-counting chamber the cells can be rapidly enumerated. With this no immediate differential count is possible; indeed, there are few specimens that yield in the counting chamber material sufficient for differential enumeration. For this purpose another part of the fluid is well centrifugalized, decanted, and the sediment left clinging at the base of the tube taken up in a capillary pipette, spread, dried, and stained.

Joffroy and Mercier put the normal number of cells as 0 to 2 per c.mm. They consider over 5 per c.mm. as strictly indicative of a pathological process in the meninges; they deem doubtful a count between 2 and 5 per c.mm., and advise another puncture.

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[8] Compt.-rend. Soc. de biol., Paris, 1901, liii, 529.

[9] Jour. Ment. Path., New York, 1902, iii, 57.

[10] Wien. med. Presse, 1904.

[11] Amer. Jour. Insan., Utica, N. Y., lx, 107.

[12] Jour. Mich. Med. Soc., 1906, v, 367.

The proteins of the cerebrospinal fluid are two, serum albumin and serum globulin, a trace only of the latter being present. An increase in the protein amount during purulent meningitis, and sometimes after intracranial hemorrhage, was early noted; but it remained for Guillain and Parant, in 1903, to point out a protein increase and the individual constituents thereof, in certain chronic nervous diseases, notably paresis. They treated the fluid in the cold with a saturated solution of magnesium sulphate, boiled the filtrate, and found a considerable quantity of albumin present. They thought albumin always pathological, but Sabrazes[13] demonstrated a trace of it in the normal fluid. Nageotte advised the routine calculation of the total protein by means of Esbach's solution and a 1 to 10 dilution of the fluid. Nissl[14] uses Esbach's solution, a centrifuge tube of special shape and graduation, and he reads off the precipitate in terms of an arbitrary scale, not of one reduced to terms of grams per liter. With this apparatus Rehm has found that the average amount of protein in the cerebrospinal fluid of paretics is much above the normal, but that there are individual instances in which the upper limit of the normal is not exceeded.

So far, careful analyses have shown that the gross quantity of protein alone is important for clinical purposes. A method such as that of Esbach for the urine seems sufficient for the estimation, but Nissl's modification of this method requires a special instrument; and Nageotte's 1 to 10 dilution of the fluid gives scale readings ($\frac{1}{10}$ gram for each gram actually present) which suppose more accuracy than the Esbach tube affords. Accordingly, in my own work, a simple means was sought by which the undiluted specimen could be used. The Esbach tube commonly sold for urine work requires more fluid than is safely obtainable at lumbar puncture, and, therefore, a test-tube of approximately 9 mm. bore was adopted. From it readings were taken as from the Esbach, and it became clear on experiment that such readings correspond with those obtained in the wider tube. Fortunately the precipitate from specimens that are normal is slight. In the markedly pathological the narrow tube sometimes chokes with flocculi. Fluids supernatant after centrifugalization of the cells may be employed for the test: 5 to 6 c.c. is required. In the cases here given the specimen was not acidified before addition of the picric-acid solution, though the reaction was, when tested, faintly alkaline to litmus, as is the rule with cerebrospinal fluid obtained during life. It may be objected that the method is arbitrary until proved otherwise, that it is coarse, and that it is open to well-known errors, dependent on temperature changes, and on the presence of much protein; but it suffices for the purpose in hand. It was used for the cases that follow.[15]

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[13] Gaz. hebdom. des Sci. méd. de Bordeaux, 1903, 376 (reviewed in Rev. neurol., Paris, 1903 xi, 1062).

[14] Centralbl. f. Nervenh. u. Psychiat., Coblenz and Leipzig, 1904, xv, 225.

The pressure in the subarachnoid space in health equals that in the ventricles of the brain; and the pressure found on lumbar puncture thus indicates that of the fluid in the whole central nervous cavity, unless there exists an obstruction of some sort. Krönig defined the normal pressure, with the patient in the lateral decubitus, as between 120 and 180 mm. of distilled water. The difficulties of exact measurements are obvious, so that most investigators are satisfied with stating that the pressure, as determined at lumbar puncture, is high or low, according as the fluid spurts or drips slowly from the hollow needle. Any tissue shred which prevents quick escape of the fluid has a power to nullify entirely an observation of this sort: and not infrequently it has been found that, though the fluid flows slowly, the pressure is high. For measurements to be precise none of the fluid should leave the subarachnoid space, since with its outflow tension is at once lowered. But for clinical measurements it is indifferent that 1 to 2 c.c. of the total 120 to 150 c.c. (Mott) should escape. One may, then, join a vertical tube directly to the puncture needle, and thus estimate pressure in terms of the fluid itself. This method for several years has been in use at the Johns Hopkins Hospital. The tube employed is thick-walled, of approximately 1.5 mm. diameter, and it has a capillary error of 20 mm. of normal cerebrospinal fluid. To measure a pressure of 500 mm. in such manner requires only 0.9 c.c. of the fluid. Pulse and respiratory waves in the column show its direct pressure connection with the subarachnoid space. When these are lacking a technical error may be looked for.

With the means described, fluids from the following cases have been examined:

Group I. Cases of disease other than nervous.

Group II. Cases of nervous disease, showing no abnormality of the fluid.

Group III. Cases of tuberculous meningitis.

Group IV. Cases of "purulent meningitis" due to infection with the streptococcus, Diplococcus pneumoniæ, or Micrococcus meningitidis.

Group V. Cases without "purulent" or tuberculous meningitis, but with abnormality of the cerebrospinal fluid.

Properly speaking, only fluids from Group I may be considered as normal—merely normal, in the sense that they were not associated with a meningeal lesion, for the patients were often very ill and not infrequently delirious. In Group II the fluids bore the characteristics made familiar by those in Group I, that is to say, the findings seemed void of any evidence for a meningeal lesion. One may, then, in a discussion of what constitutes the clinically normal fluid, take Groups I and II together. It must be admitted that in the instances in which there was no autopsy the absence from the meninges of a pathological process cannot be absolutely affirmed. In these instances, however, the course of the disease subsequent to lumbar puncture did not suggest such process. Whether the meninges can be injured without evidence of it in the fluid at one time or another is a point only recently suggested and as yet unsettled.

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[15] Doubtless a solution of phosphotungstic acid in hydrochloric acid, such as that devised by Boggs for the estimation of the protein in milk (Johns Hopkins Hosp. Bull., 1906, xvii, 342), would, by reason of the even precipitation it causes, prove better than Esbach's solution in dealing with the cerebrospinal fluid.

Group I. Cases of Disease other than Nervous.

Nine specimens of fluid were examined from 7 patients, of whom 2 had acute lobar pneumonia, 1 general tuberculosis, 1 tuberculous peritonitis, 1 arthritis deformans, 1 amœbic dysentery, and 1 a pelvic abscess. In the 3 cases that came to autopsy the meninges were normal.

Group II. Cases of Nervous Disease, Showing no Abnormality of the Cerebrospinal Fluid.

From the 7 cases included here 11 specimens of fluid were obtained. These came from an arteriosclerotic with mental symptoms, from 2 patients with tabes dorsalis (the disease of ten and thirty years' duration respectively), from 1 case of brain tumor, 1 of Korsakoff's psychosis, 1 of internal hydrocephalus, and 1 of Little's disease. Two autopsies were obtained, at each of which the meninges were found to be normal.

The number of cells in the 20 instances varied from 0.5 to 6.5 per c.mm., with an average of 2.75. In no specimen figuring here, or in the groups that follow, were cells entirely absent, though they were often few; and it seems doubtful whether they are ever quite lacking, as many think. The fact that the fluid on standing even four hours suffers a diminution in cells, and in the course of forty-eight hours often loses them completely, may explain such opinion. The upper limit of the normal, as given by Joffroy and Mercier, Fuchs and Rosenthal, may also be called in question. The case of pelvic abscess in Group I showed (at first puncture) 6.5 cells per c.mm., without at autopsy any perceptible meningeal change.

On a puncture in one of the instances of acute lobar pneumonia the fluid was contaminated with blood. In such instances the value of a counting medium which preserves the red cells becomes apparent. Bard, studying the tonicity of the cerebrospinal fluid as indicated by its behavior to the red cells, has shown them to be preserved in the normal and in some pathological fluids. Should the fluid be hypotonic and the blood laked, as may happen on centrifugalization, instead of a colorless fluid above the sedimented corpuscles, one tinted with hemoglobin is found. In the case cited 28 white cells per c.mm. were present with many red cells. The latter could also be counted, since the fluid was hypertonic: they were 20,000 per c.mm. From the ratio between the erythrocytes (4,700,000) and the leukocytes (6800 per c.mm.) in the patient's circulation on the day of puncture, it could be said that the increased number of white cells in the cerebrospinal fluid was due wholly to blood contamination. A similar process was required for one specimen of the five taken from the case of Little's disease in Group II, and here also the fluid was found normal as regards cells. Such conclusion is safe. It is a very different matter to call pathological a fluid obscured by fresh blood, on the ground that many more white cells are present than the blood seems to account for. Here one must be quite sure that no laking has occurred. Laking may be detected from the tint of the fluid supernatant after centrifugalization or from the aspect of the red cells in the counting-chamber, which appear as "shadows." Further on will be considered a specimen which, though contaminated with blood, could be determined to be pathological. Thus a fluid rendered bloody at the time of puncture may always, in case no laking has taken place, be used for a cell count. A differential count of the cells intrinsic to the fluid is, of course, impossible under the circumstances.

From Groups I and II eight differential counts were made with Hastings' stain. Mononuclears of all sizes up to those which may be classed as "large" were seen, but no polymorphonuclears were observed except in association with blood.

The protein content varied but little, considering the method employed; the lowest amount was $\frac{1}{6}$ gram per liter, the highest $\frac{3}{8}$ gram, the average $\frac{1}{3}$ gram. These figures agree roughly with those from careful analyses. Even at this date there is a wide variation in the latter. Halliburton (in a patient with escape of cerebrospinal fluid through the nose) found 1 gram per liter, and Nissl quotes the findings of various observers as follows: Quincke, 0.2 to 0.5 gram per liter; Riecken, 0.5 to 1 gram per liter; Gumprecht, 0.25 gram per liter. For clinical purposes, the method gives, in a most general sense, a quantitative determination of the normal amount of protein. With it this averages $\frac{1}{3}$ gram and is always below $\frac{1}{2}$ gram per liter.

Pressures varied between 70 and 300 mm., with the high average of 210 mm., a considerable variation from Krönig's "normal range" between 120 and 180 mm. But it is to be remembered that any factor altering the blood pressure, such as coughing, straining, or fright, affects the pressure in the subarachnoid space. Position of the body affects it too: hence all pressures were taken, as is desirable, with the patient lying on his side, the knees drawn up, and the head on a level with the vertebral column. The one point to be emphasized is that the pressure varies much more than observations on a sound person, under special conditions, would lead one to suppose. It is unwise to infer anything awry in the central nervous system from pressure changes, except when they are most marked.

Group III. Cases of Tuberculous Meningitis.

There were 9 of these and in all 17 punctures were made on them. Two of the cases lacked verification by autopsy or by the discovery of acid-fast bacilli in the fluid, but clinically they were characteristic.

In 5 of the patients the fluid gave evidence of the existence of a pathological process—by color, by haziness, or by the formation of a reticulum on standing; but in 4 there was nothing macroscopically to denote it, in spite of the presence of as many leukocytes (in one instance) as 300 per c.mm. Froin and Ramond[16] have found in a clear cerebrospinal fluid as many as 500 white cells per c.mm.

In all of the 17 specimens of fluid the cells were much increased. Never below 43 per c.mm., the count ran as high as 686 with an average of 213; and in all cases and at all punctures but one there were over 100 per c.mm. Fuchs and Rosenthal, Froin and Ramond give the only other series of quantitative determinations for tuberculous meningitis. In the 7 cases examined by the former investigators the cells numbered from 80 to 952 per c.mm. Froin and Ramond found a greater variation in their 24 specimens from 9 patients; there were "according to the cases, from 5, 16, 20, to 80 white cells per c.mm. to 800, 1000, and 1200." These authors unfortunately do not state whether the low counts were obtained early in the course of the disease, and whether the pathological findings giving rise to them were unusual. In any event it is clear that from among 48 specimens of cerebrospinal fluid yielded by patients with tuberculous meningitis only 4 contained less than 80 white cells per c.mm. and only 1 of these gave—under circumstances of which we do not know—a normal count.

In this connection may be cited the few counts on record for other diseases, the results combined of Joffroy and Mercier, Rosenthal and Fuchs, Kramer, and Simpson:

Paresis: 80 cases—5 to 204 cells per c.mm. (lymphocytes: in only 6 cases were over 100 cells per c.mm. obtained); 9 cases—0 to 5 cells.

Dementia Præcox: 25 cases—0 to 2 cells.

Tabes: 4 cases—17 to 88 cells; 4 cases—0 to 5 cells.

Alcoholism: 17 cases—0 to 2 cells.

Miscellaneous Psychoses (not on a syphilitic basis): 24 cases—0 to 2 cells.

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[15] Compt.-rend. Soc. de biol., Paris, 1905, lix, 417.

In summary, the fluid in paresis has in nearly 90 per cent. of the cases an increase in cells, while in dementia præcox, in alcoholism, and in psychoses which do not depend on syphilis the count is normal.

A comparison of the figures for tabes and paresis with those for tuberculous meningitis makes it plain that in this last disease the "spinal leukocytosis" is most constant and most pronounced. The mass of cases show more than 100 cells per c.mm., whereas it is the rule in paresis that the cells, while increased, range well below 100 per c.mm. The 3 cases of cerebrospinal syphilis in Group V agree with this rule.

That the precise count has little value when the cell increase is large was brought out by collecting the fluid at one puncture in three tubes, and estimating the cells from the first and last of these. A difference, often great, between the two was demonstrable: Tube I, 325 leukocytes per c.mm., Tube III, 200 leukocytes; Tube I, 266 leukocytes, Tube III, 686 leukocytes. Cases in Groups IV and V also showed this. Sometimes the first tube had more cells per c.mm., sometimes the last, but it is interesting that, whichever the arrangement, it held good through later punctures on the same patient. Doubtless such a relation has some bearing on the site of the lesion, and the thought at once suggests itself that if the patient be in the sitting posture when tapped the comparison of the first with the last fluid in special cases might give some clue as to whether the lesion is generalized or is more marked high or low in the spinal column. But in any attempt to secure enough fluid to yield a contrast between the first fluid and the last drawn off the danger of the procedure must always be borne in mind; the subarachnoid space must not be more depleted than is safe.

The differential counts brought out little beyond the general tendency to lymphocytosis. Cell percentages were the same in the first fluid and in the last obtained at the same puncture. No change in the count common to all cases as they progressed was noted. Indeed, the fallacy of other than the most general cell formulæ has now been recognized by Widal and Sicard, who were themselves responsible for the doctrine of specific formulæ. To quote them: "The presence of polymorphonuclears in the course of tuberculous meningitis coincides neither with the state of the malady, the age of the patient, the anatomical type of the meningitis, nor with the presence of secondary infective agents. There is too much discord in the cases observed for one to establish a rule."

Throughout the series the protein content was increased. It ranged from 0.5 gram per liter to, in one instance, over 10 grams. This last case was exceptional in many ways and the autopsy showed an extraordinary gelatinous meningitis. In the other 8 cases, which clinically or pathologically approached the type, the protein did not measure more than 1.75 gram per liter and averaged slightly less than 1 gram.

Pressure at the initial puncture varied between 240 and 550 mm. of the fluid itself, with 365 mm. as an average. Later punctures usually showed a lower pressure, once 50 mm., so that the extremes of pressure were 50 and 550 mm. The mean, greatly above that for the normal, was 308 mm. for the 13 instances in which pressure was measured.

Most importance attaches to a view of the class as a whole. Protein, pressure, and cell-content were increased largely, even in specimens quite limpid. The results as regards cell-content were more marked than those in other processes—paresis, tabes, syphilis—yielding lymphocytosis in a clear fluid. While a diagnosis of tuberculous meningitis *intra vitam* cannot be absolute without discovery of the characteristic bacilli or a positive result on animal inoculation, yet a clear or slightly turbid cerebrospinal fluid under increased tension, with over 100 cells per c.mm., among which lymphocytes predominate, and an increase in protein on Esbach's test, strongly point to the lesion. A long-standing purulent meningitis in which the many polymorphonuclear cells have been replaced by a few mononuclears, and an occasional case of paresis, are the only other conditions likely to yield such a fluid.

The power to confirm, by these means, a diagnosis of tuberculous meningitis is not so important as is that to rule it out absolutely. As has been shown, there were, in all the specimens that came under my observation, readily appreciable changes in the fluid as regards pressure, protein, and cell number. In several other patients tuberculous meningitis was suspected at the time of puncture, but results from the fluid seemed against this and the subsequent course of the cases proved other maladies to have been present. Mere failure to find tubercle bacilli in the cerebrospinal fluid does not enable one to dismiss with certainty the possibility of tuberculous meningitis, nor does the finding that cells in the fluid are few by the method of centrifugalization and an estimation of cell number per oil-immersion field. But it would seem that one can affirm the absence of the disease when the fluid conforms to the normal as regards pressure, protein, and cell-content, as here determined. Whether there are exceptions to this remains to be observed.

Group IV. Cases of "Purulent Meningitis" due to Streptococcus pyogenes, Diplococcus pneumoniæ, or Micrococcus meningitidis.

Twelve punctures were made on 6 cases—1 of streptococcic meningitis secondary to otitis media, another developing in the course of an ulcerative endocarditis due to the streptococcus, 1 of pneumococcic meningitis following operation, and 3 due to Micrococcus meningitidis. It may be urged that determinations of the sort dealt with here are scarcely necessary, since immediately on puncture the condition of the fluid is obvious. Indeed, from several specimens a coagulum separated so rapidly as to prevent the cell count. Some cases in which this occurred are not included in the group.

During the acute process, so far as the cases I have studied may indicate, pressure, protein, and cell-content of the fluid are all high—protein and cell-content higher usually than in tuberculous meningitis. Among the cells present polymorphonuclear neutrophiles predominate. A typical finding is that in the case of J., obtained at the second day of a streptococcic meningitis following otitis media: pressure 380 mm. of the fluid itself, protein 4 grams per liter, leukocytes 4700 per c.mm., 96 per cent. of them neutrophiles. Pressure in the acute cases ranged between 290 and 560 mm., averaging 390 mm.; protein ranged from 1 gram to 5 grams and averaged 2.5 grams; and the cells varied from 1300 per c.mm., 20,000, 30,000, to such numbers as made up a thick pus.

With chronicity came mononuclears and a fluid that could not be distinguished by protein, pressure, cell number and character from that of tuberculous meningitis. To cite an instance: A specimen from B., in the sixth week of cerebrospinal fever, registered a pressure of 230 mm., protein 1 gram per liter, cells 580 per c.mm., of which 67 per cent. were lymphocytes, 1.5 per cent. large mononuclears, 31.5 per cent. neutrophiles. It is also questionable whether a distinction from tuberculous meningitis can be made when a process, later to become purulent, is slowly gaining headway, or when a purulent lesion is focal in character. A patient with ulcerative endocarditis was delirious when first punctured, but presented no other symptoms suggesting a lesion of the central nervous system. At this time pressure and protein were normal, but a cell count of 15 lymphocytes per c.mm. was thought to indicate a slight meningeal reaction. Four days later, just before death, he developed stiffness of the neck, but without Kernig's sign or other suggestive feature. Puncture yielded, under a pressure of 290 mm., a purulent fluid, sterile to culture, which had 5 grams of protein per liter, and 8400 white cells per c.mm., of which 98 per cent. were neutrophiles; and autopsy showed an ulcerative endocarditis (streptococcic), infarct of the spleen, and a purulent meningitis, localized almost completely at the base of the brain. In this case one could infer no further from the first puncture than that slight meningeal irritation seemed present.

The first few cubic centimeters and the last, obtained at one puncture, frequently differed much in cell number, just as in tuberculous meningitis. Sometimes this was noted macroscopically when no count could be made.

Group V. Cases without "Purulent" or Tuberculous Meningitis, but with Abnormalities in the Cerebrospinal Fluid.

These deserve an almost individual consideration.

There were 3 instances of brain tumor and 4 punctures made on them. At these the pressure was found to be, as is usual, high—380, 490, and 580 mm. (and once not measured exactly) on estimation as previously described—but in only 1 case was the fluid itself pathological in the sense considered here. In this patient, a man with cerebral tumor, for which later a decompressive operation was performed, it was amber yellow, with 1 gram protein per liter, and 5.5 white cells per c.mm., 83 per cent. of them lymphocytes, 9 per cent. large mononuclears, and 8 per cent. polymorphonuclear neutrophiles. In Group II was cited an instance of brain tumor in which protein, pressure, and cell-content were entirely normal. Babinski and Nageotte found an absence of white cells from the cerebrospinal fluid in 7 cases of cerebral tumor, and Widal in 9; Laubry, using the same technique, which was to ascertain cell number per oil-immersion field in the sediment obtained by centrifugalization, observed a slight lymphocytosis in the fluid obtained from a patient with endothelioma of the cerebellum. These results do not permit generalization with regard to the findings in the cerebrospinal fluid of brain tumor, further than a remark on the frequent incidence of increased pressure in the subarachnoid space, as already noted at lumbar puncture by many observers.

One case of uremia exhibited high pressure (350 mm.) without cell or protein increase. Another, B., had uremic convulsions with hemorrhage into the brain six days previous to the first tapping. This gave a fluid contaminated with fresh blood: pressure 590 mm., protein $\frac{1}{3}$ gram per liter, cells—Tube I, erythrocytes 14,300 per c.mm., leukocytes 99; Tube III, erythrocytes 24,280, leukocytes 275. At a second tapping twenty-four hours later, pressure was 420 mm. of the fluid itself, protein as before, and cells—Tube I, erythrocytes 29,200, leukocytes 578; Tube III, erythrocytes 21,600, leukocytes 817. At both the fluid was hypertonic; correction for the leukocytes introduced with the blood could be made on the ratio of the two in the blood as previously explained; and it became evident that many more leukocytes were present than a contamination could account for. So the fluids were deemed pathological as regards cells. A puncture thirteen days later helped confirm the observation; for the fluid now limpid and at a pressure of 300 mm. had 12 white cells per c.mm., lymphocytes this time, whereas before polymorphonuclear neutrophiles had predominated.

H., a baby aged two months, had convulsions in association with otitis media. A puncture at the height of the illness showed 47 white cells per c.mm., all neutrophiles. Cultures were negative.

The cerebrospinal fluid of the healthy baby has not been found to differ as regards cells from that of the adult; it may reasonably be inferred, therefore, that this patient had a slight meningitis.

Two specimens were examined from a case of syphilitic insanity, 1 from an instance of syphilitic hemiplegia, and 1 from a patient with chronic spinal meningitis, enduring twelve years, who denied venereal disease, but gave a history of sores, without varices, on both legs five years previous to his admission to the hospital. All showed cells in considerable numbers (45, 64, 45, 36 per c.mm.—lymphocytes exclusively) and two increased protein (a specimen from the first case $\frac{5}{8}$ gram per liter, that from the last $\frac{2}{3}$ gram). It is to be noted how far short falls the number of cells, 48 per c.mm. on the average, of the number found in tuberculous meningitis. The pressure, too, was only slightly or not at all increased in these cases (240, 50, 190 mm.). Sicard and Monod, and many workers after them, have noted a lymphocytosis in the fluid in cerebrospinal syphilis, but there are unfortunately no figures to indicate its degree. It seems, though, from these few observations to approximate that met with in paresis. The idea has been put forward that in paresis, tabes, and cerebrospinal syphilis the degree of lymphocytosis in the cerebrospinal fluid has relation to the acuteness of the process. The 2 cases of tabes entered perforce in Group II, because the fluid from them was not abnormal, had lasted ten and thirty years respectively, and the disease had ceased to progress.

Two hydrocephalic children showed high pressure (Case W., 560 mm.; Case G., 320, 500, and 150 mm. at the three punctures), with cells and protein at normal. One served to demonstrate a fact of much import. This patient, G., had some fluid withdrawn on three occasions at forty-eight-hour intervals. At first puncture the cells were 2 per c.mm.; at the second 183 per c.mm., mostly neutrophiles; at the third 18 per c.mm., largely lymphocytes.

Apparently the initial puncture gave rise to a meningeal irritation of some sort (whether bacterial is not known, as no cultures were taken) and this underwent rapid subsidence. The child's condition did not vary with these changes. On the second puncture a trace of fresh blood was found in the fluid, which itself remained yellow after centrifugalization. The red cells seen were well preserved, but from the tint of the fluid one might suppose partial laking to have occurred and attribute the whole increase in white cells to contamination, were it not for the preponderance of mononuclears at third puncture (in the clear fluid from which a few red cells were also obtained). The entire change in cell formula thus shown could hardly have taken place in the circulating blood during forty-eight hours, and this with no variation in the child's condition. On the other hand, such a change of the leukocytes present in the subarachnoid space is common. Assuming that this occurred, it is not improbable that an oozing of blood from the first puncture wound into the cerebrospinal fluid, there breaking down, accounts both for the yellow color of the specimen obtained later and for its considerable content of white cells. However this may be, the theme is supported that, when the count from a second puncture coming soon after a first demonstrates a moderate increase in cells over the normal to be present, there is a possibility that this cell increase depends solely on injury done at the first puncture. The book rule to wait ten days before a second puncture seems justified.

Under such reasoning the result in a case of toxic dementia, which falls in Group V because of a cell increase (cells 16 per c.mm.) is vitiated, and so too with the findings at second puncture of the patient with pelvic abscess in Group I (cells 11.5 per c.mm.). In each of these cases the subarachnoid space had been tapped shortly before.

Three cases remain undiscussed: In one, a toxic psychosis, the protein content was increased ($\frac{5}{8}$ gram); in the second, of general peritonitis, the pressure was equal to 400 mm. of the fluid; and in the third, the cerebrospinal fluid was abnormal, but the disease was, and remained, undiagnosticated. It may be thought that the findings in the first 2 cases should be placed with those obtained in Groups I and II, as the one case recovered without signs of meningitis, and the other had at autopsy no recognizable central nervous lesion. But, as the first was delirious and the second in coma at the time of puncture, it is safer to take them as unexplained variations rather than to allow them to modify one's conception of the normal fluid. Statistics must determine whether the range for the normal includes them.

In the differential counting for Group V, as well as in that for the other groups, there were frequently noted bodies, apparently organic, which could not be classified as cells and were not included in the counts made. Reticulated, basophilic, varying much in outline, they were the so-called "degenerated cells," "clear elements," "cellular transformations" which are sometimes mentioned. Villaret and Texier,[17] who alone seem to have given thought to the origin of these, separate three sorts derived respectively, they think, from the small mononuclears, from the large mononuclears and the polymorphonuclears, and from the endothelial cells. Though they have not supported their opinion experimentally it should be borne in mind, for the presence of these elements, if degeneration products, in large numbers, with but few normal cells, might indicate the waning of some previous meningeal inflammation. The autolytic digestion of leukocytes is now well understood; and there seems nothing against self-digestion of those in the cerebrospinal fluid (which itself contains no enzymes), provided they be not too speedily hurried into the lymph stream.

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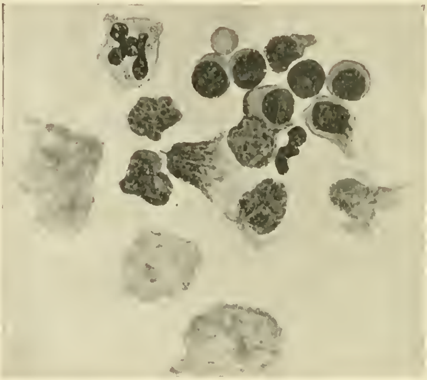
[17] Jour. de physiol. et de path. gén., Paris, 1905, vii, 841.

An attempt was made, in my work, to show whether the elements under discussion are degenerated cells. For this purpose specimens, obtained aseptically, of fluid known to contain many cells were incubated for varying periods and centrifugalized; slides from the sediment were spread and mounted in the usual manner. Control slides were always made from another portion of the same specimen while fresh, and cultures were taken after the incubation to demonstrate that bacteria had no part in the work. Several clear fluids from instances of tuberculous meningitis and one of normal cell-content were used. The result was in every instance the same: the cells were rapidly transformed into those doubtful bodies which had been observed on routine count. After four hours of incubation most of the lymphocytes (as lymphocytic fluids alone were used) had lost, as shown with Hastings' stain, the distinction between nucleus and cytoplasm, were larger, slightly irregular in outline, and a deep bluish-purple with light splotches here and there. After eighteen hours all the cells were replaced by elements, the better preserved of them 15 to 25 μ in diameter, reticulated, basophilic, sharply defined at the edges, without trace of a nucleus. From these all gradations could be followed until only a ragged neutrophilic film, fading at the edges, was left about a poorly staining, refractile mass, suggesting nuclear material.[18] At the end of ninety-six hours these still persisted. Incubation was not carried farther. Hastings' mixture and Löffler's methylene blue were the stains used, and the sediment from the centrifugalized fluid was spread, dried at room temperature, and stained, without intermediate pro-



Cellular content of the cerebrospinal fluid from a case of tuberculous meningitis, showing several mononuclear cells, a polynuclear leukocyte, and a number of degenerated cells. × 660. (Camera lucida drawing; Hastings' stain.)

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[18] Rubinato (Folia Hæmatologica, ii, 781), in a study of the forms occurring in the destruction of leukocytes, has very well described these appearances. He found them as the products of the degeneration of mononuclear cells only.

cedure. Violent centrifugalization and a strong stain brought out distortions of the degenerated cells, which under gentler methods could be avoided. But that such methods were not the cause of the forms described was shown by the controls.

A possible value for the fact that these "clear elements" are degenerated cells rests, as has been said, upon their significance when they make up the bulk of forms in a specimen. Can one suppose a long-standing "spinal lymphocytosis," or one previously acute, now retrograding? Unfortunately the hypothesis was too recently formulated for many instances bearing on it to be available for this paper. Certainly in the normal fluid they are always present, and in considerable number as compared with the sound cells. This is to be expected, since the persistence of one of these derivatives is longer, probably, than that of the leukocyte itself. Furthermore, in acute processes the number of them is few as compared with that in long-standing disease. They are rarely seen in smears from a purulent fluid, though in such the polymorphonuclear neutrophiles are often vacuolated and show pyknotic nuclei after the meningitis has progressed for a time.

Three cases especially showed these "clear elements": One in Group V, of "chronic spinal meningitis" with long-standing nerve-root pains, in which, at operation, a patch of thickened meninges was found, furnished them to the exclusion of the usual cells; one in Group III, with tuberculous meningitis of three months' duration, had them in large quantity; while at first puncture in a third case, that of L., described in Group IV, although the meningitis was just beginning, they were exceedingly numerous. One can, of course, offer, in this last instance, the explanation that a chronic process might previously have existed. Further work must show whether a clinical inference can be drawn from the presence in the cerebrospinal fluid of numerous degenerated cells.

My studies lead to the following conclusions:

1. The methods described herewith offer the means for a much more careful clinical study of the non-purulent, cerebrospinal fluid than has heretofore been usual. Determinations should always be made of the pressure in the subarachnoid cavity, of the protein content of the fluid, the number of cells per c.mm., and their kind.

2. By these means a specimen may often be pronounced pathological which under the ordinary examination appears normal. Admixture of blood does not necessarily render a fluid valueless.

3. Normal fluids differ but little in quantitative cell-content. It is doubtful whether cells are ever wholly lacking, and up to 7 per c.mm. may provisionally be set as the range in specimens not pathological. The normal protein-content, too, lies within well-defined limits. On the other hand, pressure variations which depend on factors other than a lesion of the central nervous system are wide.

4. Except along the broadest lines there is nothing specific in the

permutations of protein, pressure, and cells. But along these lines the fluid from cases of tuberculous meningitis is characteristic, as compared with that from cases of paresis or of cerebrospinal syphilis, and these also differ from the normal fluid. If a case be suspected to be one of tuberculous meningitis, but the fluid prove to be normal as regards pressure, protein, and cells, the disease may be ruled out. This rule probably holds absolutely, but it is desirable that more cases be studied before the statement is too strongly made.

5. The "clear elements," "cellular degenerations," "pseudo-endothelial cells," often noted, are definitely proved to be degenerated cells.

Most of the cases studied were from Dr. Barker's wards. My thanks are further due to him for the suggestion of this work, to Dr. Cushing for fluid from patients with hydrocephalus, and to Dr. R. I. Cole for much helpful advice during the work.

TROPICAL NEURASTHENIA AND ITS RELATION TO TROPICAL ACCLIMATION.

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It is strange that the nerve exhaustion of the tropics, which is so very prevalent among those who have sojourned for any considerable time in our island possessions, is scarcely mentioned by writers on tropical subjects. This form of nerve exhaustion, not so much on account of difference of symptoms from the usual manifestations of neurasthenia, but on account of its etiology, should receive the designation of tropical neurasthenia. The literature on the subject is very meagre. I cannot find mention of the disease by German, French, or English writers, and it is scarcely recognized by American authors.

Woodruff[1] states: "I have never seen any account of the terrible nervous exhaustion which results from long-continued exposure to great heat and moisture. It is a veritable neurasthenia. There is no escape from the heat, day or night. One wakes up at night drenched with perspiration. It wears out nervous force quickly and surely. Now the symptoms of exhaustion are everywhere. I never heard so many complaints of persistent insomnia as I did in the Philippines. One young, vigorous staff officer, who seemed

[1] The Soldier in the Tropics. Phil. Med. Journ., April 7, 1900; Effects of Tropical Light on White Men.

always cool, complained to me one day that on the previous afternoon at five o'clock he found that he could not remember anything he read. When he got to the end of a sentence he had forgotten the beginning. He was completely disabled until the next morning. A surgeon informed me that before he was half through his rounds, he daily discovered that he could not remember the patients' answers, and he always had to lie down thirty to sixty minutes before the brain would again work properly. This general exhaustion, which is such a common occurrence as almost to merit the special term of tropical exhaustion, deserves the greatest attention. It reduces man's resistance to every disease, as his natural immunity is gone."

I may quote the following, from an article by myself:[2] "The majority of American women who have resided for some length of time in the Philippines suffer with what we call tropical neurasthenia. They become nervous, irritable, anemic, lose weight, suffer with neuralgia, spells of faintness, sleep poorly, and almost invariably are troubled with menorrhagia and dysmenorrhœa." In a later monograph Woodruff again refers to the neurasthenia of white men in the tropics. Indeed, the object of his monograph is to prove that these tropical exhaustions are caused by the actinic or short waves of light which are of greater intensity in the tropics. Outside the above I have been unable to find anything of importance in literature relating to tropical neurasthenia—a disease, I believe, that is not only very prevalent in the Philippines, but is also an important factor in preventing acclimation of white men in the tropics and will prevent the successful colonization of the Philippines by Americans.

If we search for the true cause of the invalidism of so many public servants in the Insular service we find that a large percentage of the 50 per cent. (as stated by Musgrave[3] as due to intestinal amœbiases) had been suffering for some time with tropical neurasthenia which had destroyed their natural immunity and made them very susceptible to amœbic infection. From experience in private practice and government service, I can say that nearly all American women and a large proportion of men who have been in the Islands one year or more suffer, at least to some extent, with nerve exhaustion. It is conservative to state that 50 per cent. of the women and 30 per cent. of the men suffer with neurasthenia to such an extent that they are in a state of semi-invalidism. These persons cannot easily concentrate their minds on their work, they suffer with loss of memory, they become easily fatigued, and they cannot do the efficient work they were formerly capable of doing. There is no doubt that directly and indirectly tropical neurasthenia incapacitates more white men in the Philippines than all other causes combined and seems to be the main hindrance to the ability of white people to become acclimated in the tropics.

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[2] The American Physician in the Philippine Civil Service. Amer. Med., April 1, 1905.

[3] Treatment of Intestinal Amœbiasis in the Tropics, Bulletin No. 18, Bureau of Government Laboratories, Manila, 1904.

Symptoms. The symptoms of the nerve exhaustion of the tropics are not unlike neurasthenia, as seen in America. There are, however, a few minor differences which I shall consider.

1. In regard to women, menstrual derangements, metrorrhagia, menorrhagia, and dysmenorrhœa are much more prevalent in the Philippines as an accompaniment of neurasthenia than in this country. It is safe to say that ninety-five out of every hundred women who suffer with neurasthenia suffer with these menstrual troubles, and the majority of these to a very marked degree. These menstrual disorders are not unknown in this country. Brower has noticed them in about 20 per cent. of neurasthenic women who have come under his notice, but they were not marked. Richard Dewey has noticed a few severe cases in neurasthenics, but believes that neurasthenia did not cause, but only aggravated, the disorder. Whether these menstrual disorders are caused by relaxation due to excessive heat and a moist climate, or to some derangement in the vasomotor mechanism due to the neurasthenic condition, is still open to question. It is probable, however, that both influences work together to produce the condition.

2. Vasomotor symptoms seem to be somewhat more prominent in tropical neurasthenia. Among the most important of these are angioneurotic œdema, which Quincke regards as a vasomotor neurosis under the influence of which the permeability of vessels is suddenly increased. Pemphigus, which is generally known to accompany changes in trophic nerves, is also quite prevalent. Localized sweating, œdemas, etc., are quite common.

3. Tropical neurasthenia responds more readily to treatment than ordinary neurasthenia. Change of climate is especially effective. Many subjects leave Manila in a most exhausted condition, but after a short stay in Japan, or a voyage across the Pacific, their condition is remarkably improved.

Etiology. As regards the etiology of tropical neurasthenia—it differs from that of the neurasthenia of this country. Overwork, worry, heredity, abuse of alcohol, certain infectious diseases, etc., predispose to tropical neurasthenia, as in America, but they do not seem to be the main causes. The etiology of tropical neurasthenia may be discussed under the following heads: (1) Manner of living; (2) the effect of certain infectious diseases, such as dengue fever, dysentery, etc.; (3) the effect of the excessive use of alcohol; (4) the effect of the rays of the sun; and (5) the effect of continuous and excessive moist heat.

1. Many Americans, on reaching the Philippines, attempt to continue the same active life that they are accustomed to in the United States. They attempt to work the same number of hours, they go out in the sun without protection, and they take no midday

siesta. When going to a new country, it is always a good plan to study the prevailing customs and not try to follow one's old mode of living under different conditions. The native Philippino works fewer hours than we do. He is careful to protect himself from the hot sun, and he invariably sleeps from 12 to 3 in the afternoon. If Americans persist in disregarding the customs of the country, and try to continue their accustomed activity, they may expect an early nervous breakdown.

2. Certain infectious diseases prevalent in the Philippines predispose to nerve exhaustion. The most important of these are dengue fever and amœbic dysentery. During certain seasons of the year dengue fever is epidemic and attacks a large proportion of the population. The ensuing prostration is often extreme and usually continues long after the disappearance of the fever. In many people an attack of dengue fever is the beginning of their neurasthenic condition. Amœbic dysentery, if long continued, is very exhausting and predisposes to or increases the neurasthenia.

3. There is great difference of opinion in regard to the effect of alcohol in the tropics. Woodruff states that "statistics of about 2800 soldiers show that the damage done to these vigorous men by the small amount of excessive drinking they indulged in was not so great as the damage done by the climate to the total abstainers. Approximately, 11 per cent. of the abstainers died, while about 3.5 per cent of the moderate drinkers, and less than 2 per cent. of the excessive drinkers died. About 15 per cent. of abstainers were invalided home, about 9 per cent. of the moderate drinkers, and about 8 per cent. of the excessive drinkers." Other observers give a directly opposite opinion—that nowhere are the effects of alcohol so detrimental as in the tropics. Some writers state that the excessive use of alcohol is one of the main factors serving to prevent the colonization of the tropics by white men. I have not been able to make systematic observations of this subject and must leave the question open to farther investigation. It is possible, however, that the moderate use of alcohol may serve to prevent intestinal infection by certain parasites, (1) by stimulating the action of the gastric juice, and (2) by its antiseptic action on the parasites themselves.

4. The actinic theory of the cause of neurasthenia in the tropics, promulgated by Major Woodruff,[4] demands special attention. This work originated as stated in the preface "in an attempt to prove or disprove the theory announced by von Schmädel, in a paper read before the Anthropological Society of Munich, in 1895, that skin pigmentation of man was evolved for the purpose of excluding the dangerous actinic or short rays of light which destroy living protoplasm." The argument runs as follows:

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[4] Effects of Tropical Light on White Men.

The actinic rays of the spectrum, also called ultra-violet or photographic rays, which are invisible rays of very short wave length, are known to cause great damage to living protoplasm. Man also suffers from the effect of these rays if he is so situated that the rays are strong enough to penetrate the skin and exert their chemical effects on the tissues. It is the pigment in the skin which ordinarily protects man from the dangerous effects of these rays. The amount of pigment in each individual is in direct proportion to the intensity of the light of the country to which his ancestors have proved their adjustment by centuries of survival in health and vigor. If, therefore, man remains in the environment to which he has become accustomed for thousands of years, his pigmentation is sufficient to prevent the penetration of the ultra-violet or actinic rays. As we go south, the light becomes more intense, first, because the amount of light per unit of surface increases, and, second, as we go nearer the equator the layer of air which the rays must pass through is thinner and fewer rays are absorbed. The inhabitants of the tropics, therefore, in order to counteract these increased actinic rays have, after many centuries, developed more pigment in their skin than northern races. The white man in going to a tropical country like the Philippines leaves the environment where perhaps his ancestors have resided for centuries, and exposes himself to actinic rays which penetrate the skin (it being insufficiently pigmented) and exerts certain chemical action on the tissues which is responsible for the neurasthenia as seen in the tropics.

Woodruff has noticed that brunette types do better in the Philippines than blondes. He even contends that the entire United States, with the exception of parts of Washington and Oregon, is too far south for blondes. City life in the United States on account of the glare is especially prejudicial to the health of blondes. He cites the fact that the Norwegians and Swedes coming from 60° and above to 45° and under, arrive here as vigorous men and women, but in a generation or two they degenerate into weak and neurasthenic men and women, many of whom succumb to tuberculosis. To prove his theory, Woodruff goes into the question very fully; actinic therapy is reviewed up to date. Many examples are given illustrating the effects of excessive light and the result of the migrations of blonde races is gone into from the earliest time until now. Just how the actinic rays on penetrating the skin bring about the state of nerve exhaustion is perhaps left a little indefinite, but overstimulation seems to be the view put forth.

Dr. James Weir, Jr., is quoted as follows: "Their action (the violet rays) must be, I take it, primarily through stimulation of the vasomotor nerves. The first effect is dynamic in character, there is a dilatation of the bloodvessels with a consequent flow of blood. The second effect is chemical in nature; the increased flow of blood incites the blood-producing organs to manufacture new blood cells.

There is also increased oxidation and oxygenation through the lungs. The third effect is purely physiological. Owing to the increased flow of new blood cells to the tissues, cell growth is excited and new tissue is formed." A little stimulation like the above is considered beneficial, but as the short rays become more intense, stimulation of metabolism is increased to such an extent that the functions become exhausted and a neurasthenic condition develops.

This theory, as presented by Major Woodruff, is very convincing, and, although the effect of the violet and ultra-violet rays may not play such an all-important part as he suggested, there can be no doubt that they play an important part in the etiology of tropical nerve exhaustion.

5. As regards the action of continuous moist heat in the production of neurasthenia, we know that excessive bodily heat is regulated mainly by the skin. The more blood that passes through the skin the greater the loss of heat by conduction, radiation, and evaporation. To accommodate a great flow of blood through the skin the arterioles of the skin must dilate. This may be produced in two ways: (1) Blood of a higher temperature than natural flowing over the vasomotor centres will stimulate the vasodilator fibers and cause the arterioles to dilate, and (2) warmth acting on the exterior of the body by stimulating sensory nerves will cause the skin arterioles reflexly to dilate. Sweating usually accompanies the increased blood flow through the skin, as the sweat centres in the spinal cord are stimulated either directly or reflexly in the same manner as the vasomotor centres.

The climate in the Philippines is such that during a greater portion of the time conditions are present which excite an increased flow of blood through the skin. These conditions are present nine months out of twelve and eighteen hours out of the twenty-four, and are dependent on the fact that in the Philippines there is a high temperature and a relatively high humidity. The reports of the Manila Weather Bureau covering a period of over twenty years show that the highest mean monthly maximum temperature is 96.98° F. in May, the lowest mean monthly maximum 89.78° F. in Decemer, with an average of 92.48° F., as the average mean maximum for the year. During the same period the highest mean monthly minimum is 72.86° F., also in May, and the lowest mean monthly minimum 64.22° F. in January, with an average of 69.08° F. as the average mean minimum for the year.

The temperature in Manila not only averages higher throughout the year than in our hottest summer months, but the relative humidity is considerably higher, as seen by the following table, in which the average temperature of three American cities for the three hottest months of the year, June, July, and August, is compared with the average temperature of Manila for the whole year and with the average temperature of the three coldest months in Manila, December, January, and February.

	Mean.	Mean max.	Mean min.	
Manila	80.42	92.48	69.08	Average temperature.
Manila	77.54	90.26	64.52	Three coldest months.
Chicago	68.1	74.9	61.8	Three hottest months.
Washington	73.1	84.2	63.3	Three hottest months.
New Orleans	82.1	91.4	75.4	Three hottest months.

The effect of the temperature is still more intensified by the fact that the relative humidity is very high in Manila.

The following table taken from the report of the chief of the Manila Weather Bureau conveys an idea of the relative humidity of certain cities in the United States as compared with Manila:

	Mean relative humidity.
Manila	79.4
Phœnix, Arizona	38.7
El Paso, Texas	38.8
Denver, Colorado	48.7
Pierre, S. D.	64.7
St. Louis	69.5
Philadelphia	70.3
St. Paul	71.9
Washington	72.3
New York	73.2
Milwaukee	74.9
Chicago	74.8
New Orleans	77.3
San Francisco	79.9
Block Island	81.2
Atlantic City	81.4

There are but few localities in the United States which surpass Manila in relative humidity. Even the locality of Milwaukee and Chicago, which is considered of high humidity, is surpassed by Manila nearly 5 per cent. The effects of such high temperature combined with the high percentage of humidity is well expressed by Prof. Dexter,[5] of the University of Illinois: "Excessive heat, together with great humidity, forms a most deadly combination for one not acclimated to it."

Stewart[6] states: "In a warm atmosphere the skin is flushed with blood, its vessels are dilated, its temperature is increased; an effort, so to speak, is being made by the organism to maintain the difference of temperature between its surface and its surroundings, on which the rate of heat loss by radiation and conduction depends. . . . In dry air the sweat evaporates freely and cools the skin, while in moist air, although according to Rubner, the loss of heat by radiation and conduction is increased, the loss of heat by evaporation of sweat is diminished in still greater degree. In saturated air at the body temperature no loss of heat by perspiration or by evaporation from the pulmonary surface is possible."

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[5] Weather Influences.

[6] A Manual of Physiology.

Landois[7] states that a man is just able to work in an atmosphere at a temperature of 31° C., and almost completely saturated with watery vapor. Crombie states that Europeans in Bengal have an average of 0.41° more temperature than at home.

From these facts what must be the condition of the physical organism in the Philippines is apparent. The high temperature which is prevalent most of the time causes dilatation of the arteries of the skin with sweating. On account of the high relative humidity, evaporation from the skin is prevented to a considerable extent, often to such a degree that the organism is unable to keep the temperature down to normal. The resulting increased temperature of the blood still farther increases the dilatation of the arterioles of the skin and more blood flows through the skin to be cooled. This process is almost continuous, as seen by the excessive and almost constant perspiration which continues throughout the day and usually well into the morning hours.

The abnormal dilatation of the arterioles of the skin must cause a more or less marked anemia of the internal organs. There can be no doubt that such an anemia is produced. Kellogg[8] states that the muscles, the portal system, and the skin each is capable under certain conditions of retaining a large share of all the blood in the body, the skin by percussion, friction, reaction from cold and heat, especially the latter.

Signs of anemia of the central nervous system are observed very frequently in the Philippines. Vertigo and faintness are very prevalent phenomena, especially among women, and inability to remember and concentrate one's attention are often observed. Woodruff cites several instances of inability to think or remember without taking rest in the recumbent position. All such symptoms are most easily explained by the fact that the central nervous system is lacking in sufficient blood supply. The same cause may account for the gastric disorders that are so frequent. Granting, therefore, that a certain amount of anemia of the central nervous system exists, the question now presents itself, Is the anemia thus produced sufficient to cause, or to have some part in causing, the neurasthenia so characteristic of the tropics? I believe that such anemia is sufficient for the following reasons:

1. If temporary loss of memory can be induced by cerebral anemia it is reasonable to suppose that such anemia long continued may cause more or less permanent nervous derangement.

2. We know that if the various portions of the body are not supplied by a sufficient amount of blood they deteriorate or degenerate. The muscles grow small if they are not exercised sufficiently to cause

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[7] A Text-book of Human Physiology.

[8] Rational Hydrotherapy.

an increased flow of blood through them. If the laborer does not take sufficient rest after meals, a greater share of blood will remain in the muscles and digestive disturbances often follow because too little blood is allowed for the gastrointestinal digestion. Reasoning from analogy, is it not likely that the anemia of the central nervous system will cause a state of nervous exhaustion?

3. Several observers have noted that the nearer a white man approaches the size of the native, the better he seems able to resist the evil effects of a sojourn in the tropics. There are no statistics on record as to the size of men affected with neurasthenia, but from personal observation of those most markedly affected with nerve exhaustion, the large man seems to predominate.

The following quotation from a recent letter from Major Woodruff is much to the point: Speaking of neurasthenia, he says: "In my book I mention the facts that men of large musculature are more affected; since that was written I have learned of four new cases of these giants, all of whom have come back with dilated hearts and neurasthenia. In recent conversation with an Englishman from Ceylon, I learn that the big men break down, as a rule, and that the survivors who spend many years in India are, like Lord Roberts, the little fellows. I do not think there is any doubt that bigness makes one more susceptible, and that the nearer we approach the size of the native the better." The fact that large men are more susceptible to neurasthenia supports the anemia theory of tropical exhaustion.

Large men, especially those with large muscles, are more likely to suffer with anemia of the central system than smaller men, as is apparent from a calculation that I have made:

The area of the skin surface of a large man (six feet tall and weighing 175 pounds) was found to be 2677.6 cubic inches, and that of a small man (five feet, two inches tall weighing 114 pounds) 2272.8 cubic inches. In proportion to the weight of the two men, the skin surface of the large man was found to be 23 per cent. less than that of the small man. This deficiency of skin surface of nearly 25 per cent. makes it more difficult for these large men to cool off. In order to make up the deficiency more blood must be pumped through the skin or the cooling process must be kept up for a longer time. In either case it means an anemia of the central nervous system more pronounced than in men of smaller size and with a greater proportional skin surface. To force such an increased quantity of blood through the skin to be cooled, the heart is overworked. Hence, the extreme neurasthenia and cardiac dilatation seen in so many large men coming home from the Philippines.

This theory that the anemia of the central nervous system produced by excessive heat and humidity has some part in causing the neurasthenias of the tropics, has not been proved experimentally, but is presented as a plausible working hypothesis upon which may be based future study.

As regards acclimation, we cannot say that people are acclimated to a certain locality unless they can live there with comfort to themselves, their children, and their children's children. It does not follow, as some authors say, that because a certain proportion of the American people may live in the tropics with apparent comfort and good health for a certain time that acclimation is possible, and that it is possible for the white race to colonize in the tropics. After only a few years of residence in the tropics the white people become a race of neurasthenics. We know that neurasthenia is but a step toward degeneracy. What, therefore, will be the condition of the children of these people or of their grandchildren?

Born of neurasthenic parents, they will inherit an organism lacking in nerve force; being forced to live in an enervating climate, their small reserve will be still farther drawn upon, and in a generation or two there will result a race with little resemblance to the mother stock, small, puny, weak-minded, in fact a degenerate race which would soon cease to exist if new stock did not continually come from the home land.

Prophylaxis and Treatment. It is evident from what I have said that certain persons should avoid the tropics. These include blondes, large men with great musculature, those with tuberculous tendencies, those having disorders of the stomach and intestines, neurasthenics (including those suffering with headaches, insomnia, hysteria, insanity, etc.), women, children, and old men.

Many Americans contract pulmonary tuberculosis in the Philippines, and unless they change their climatic surroundings at once, they decline rapidly. The chances of contracting tuberculosis are greater than in America, in spite of the out-of-door life, because: (1) The natural resistance of the organism is greatly diminished, and (2) the disease is much more prevalent in the Islands.

In the United States, the yearly death-rate for pulmonary tuberculosis per 1000 inhabitants runs from 0.7 in the country districts, to 1.7 in some of our larger cities, while in Manila it is over 5.[9] The Philippine Civil Government has provided no hospital or sanatorium for the tuberculous sick, so that without treatment they are allowed at large, and infect the streets and their homes with their sputum.

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[9] A study of tuberculosis in the Bilibid Prison teaches a lesson as to the effect of overcrowding on this disease. This prison was originally intended for from 1000 to 1200 prisoners. In 1902 the number of prisoners averaged about 2000, and during that time the yearly death-rate from pulmonary tuberculosis per 1000 was 9.5. During the latter part of 1904 the number of prisoners was very much increased. For the twelve months ended October, 1905, the average was approximately 3900 During that year the yearly death-rate from tuberculosis per 1000 was over 52, or more than ten times the usual death-rate for that disease outside the prison. A moderate increase of pulmonary tuberculosis would be expected during confinement, but when during one year out of 3900 prisoners over 200 die of this disease something is radically wrong.

Nervous diseases are aggravated in the tropics. Insanity is much more prevalent in the Philippines than in America. In the report of the Surgeon General of the United States Army, for 1902,[10] we find that for the year ended June 3, 1901, the insanity rate among soldiers per 1000 for those at home was 1.38, while in the Philippines it was 2.79 for regulars and 3.9 for volunteers.

Old age, as well as childhood, contraindicates a sojourn in the tropics. Woodruff's statistics show that the best age is from twenty to thirty or thirty-five years. "No man over fifty-five should dare to go there except for a short visit."

Rules of tropical hygiene may be considered under the following heads: (1) The avoidance of the direct rays of the sun; (2) the avoidance of excessive heat; (3) food and its preparation; and (4) excessive labor and long hours.

1. The actinic rays may be avoided, to a certain extent, (1) by exposing one's self to the sun as little as possible; and (2) by employing proper protection and clothing. Panama hats and the Philippino straw hats afford inadequate protection for the head. When compelled to go in the sun, a black umbrella should be used, or better still, one should be provided with a broad-brimmed cork helmet which will not allow the actinic rays to penetrate.

The clothing is important. Materials should be used of sufficient opacity to prevent as much as possible the penetration of the actinic rays, but should be light enough so that the process of cooling off be not interfered with. Both theory and practice demonstrate that the outer garments should be white, since this color absorbs less heat than darker colors. The color which shows the greatest opacity to light rays is black. The underclothing, therefore, should be black and of a light material, preferably linen. During the cool evenings of the winter, flannel underclothing may be worn to prevent the too rapid cooling.

Major Woodruff calls attention to the unsuitability of the Philippine houses for Americans. Not enough provision is made to avoid the excessive light. The houses should be built with large, shady verandas, the roofs of which should slope well down, so that those sitting on the porch do not get the glare from the sky. The windows should be protected with awnings and there should be no iron roofs to reflect the heat and make the house no less than a furnace.

The midday siesta should not be forgotten. The avoidance of muscular activity for a few hours during the hottest portion of the day lessens excessive heat production; and the strong midday light rays are avoided.

2. To avoid excessive heat in the tropics is not always possible, but during the hottest months, May and June, it is advisable, indeed it is almost a necessity, that Americans sojourn in the cool regions

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[10] Report of Surgeon-General, U. S. Army, 1902.

of Japan or in Baguio, the summer capital in the mountains of Benguet.

3. The nervous exhaustion so prevalent demands the most nourishing of diets. It is a mistaken notion that dwellers in the tropics need little meat. More meat is needed than in our temperate climate. To avoid intestinal infection, all food must be aseptically prepared. Green vegetables of all kinds are dangerous, and it is doubtful if fruits even with thick coverings are safe. All water used for drinking purposes should be previously boiled, and while in a boiling state placed in retainers under lock and key until ready for use. Distilled water should not be considered safe, but should also be boiled, since a recent epidemic of amœbic dysentery in Manila was traced to an infected distilling plant.

If bottled water is used the brand must be selected with great care, as a great deal of the bottled water for sale in Manila is unreliable. The possibility of amœbic infection from ice washed with hydrant water and the rinsing of glasses in hydrant water without properly drying, must be borne in mind.

Americans must avoid excessive labor and long hours in the tropics. Manual labor in the sun is out of the question. Light manual labor in airy shops is less depressing, but should be limited to five hours daily. Mental work also is much more fatiguing than in America. Prolonged concentration of the mind is almost impossible. For the Philippine Civil Government to require more than five hours daily work by its employés means a less efficient service and an early breakdown. The statement made by some writers that American employés are able to do more work in the Philippines than at home is absurd in the extreme.

As regards treatment, early change of climate is usually all that is necessary. It is remarkable what a great change is produced in the patient's condition by a few months in Japan or a trip across the Pacific.

EXPERIMENTAL ARTERIAL DEGENERATION.

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The first successful experimental arterial degeneration should be credited to Lion and Gilbert,[1] who, in 1889, produced changes in the media by repeated intravenous injections of micro-organisms or their toxins. These changes were described as sclerocalcareous and were apparently of the same character as those produced subsequently by adrenalin. Therese and Crocq[2] later verified these results. The next successful attempt recorded was by von Eiselberg[3] who, in 1901, reported arterial changes, very similar in nature, in thyroidectomized animals. Josue,[4] however, in 1903, following his successful attempts with adrenalin, imparted the first real stimulus to work along this line. He gave rabbits daily, for a few weeks, intravenous injections of adrenalin chloride and was able to produce areas of calcification in the thoracic aorta. Macroscopically, these appeared as small depressed areas, circular in outline, the floor being thin, at times showing aneurysmal bulging or distinct aneurysms. Microscopically, as has been shown especially by Zeigler,[5] Erb,[6] and Stanton and Pearce,[7] there is primarily a degeneration of the muscle fibers in the media, the intervening elastic fibers losing their wavy outline but otherwise not undergoing any change. Lime salts are rapidly deposited in the area of muscle degeneration, the resulting rigidity of the vessel wall leading to transverse fracture of the elastic fiber. The process is thus confined primarily to the muscle fibers, the elastic tissue being secondarily involved. The adventitia and intima are not involved, except in the process of repair, when, as has been demonstrated by Stanton and Pearce, the defect in the wall is obliterated by cells arising from the intima. The degenerated process is confined chiefly to the thoracic aorta. The renal and mesenteric vessels were also involved in some of Erb's cases, and Fisher has reported a hemiplegia resulting from rupture of a cerebral vessel.

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[1] Des séances de la Soc. de biol., October 12, 1889.

Josue's results have been verified by numerous investigators and have led to experimental work with other substances in order to see whether similar vascular changes may be produced. At the present writing, more or less extensive arterial degeneration has followed the intravenous injection of digalen,[8] nicotine,[9] phloridzin,[10] and various synthetic preparations resembling adrenalin, and the administration of lead salts[11] and tobacco[12] by the mouth.

It is quite remarkable how early these changes may occur and especially the rapidity with which lime salts are deposited. Braun[13] found macroscopic changes after five injections and microscopic changes after two. Rzentkowski[14] reports calcification as early as the fifth day, and Zeigler the sixth day. On the other hand, daily injections repeated for several weeks give negative results in some cases, as there is apparently a marked variation in the individual resistance to these alterations. Apparently the age of the animal is an important factor, as Pic and Bonnamour[15] and later Pearce and Stanton found it much more difficult to produce degeneration in the vessels of young rabbits than in old ones.

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[2] Archiv. de Méd. exp., 1894, p. 583.

[3] Deutsch. Chirurgie, 1901, S. 47.

[4] Soc. de biol., Paris 1903, p. 137.

[5] Beiträge zur path. Anat. und allg. Path., Bd. lxxxiii, S. 229.

[6] Münch. med. Woch, 1905, S. 829.

[7] Jour. Exp. Med., 1906, p. 74.

[8] Deutsch. med. Woch., 1905, S. 1714.

[9] Amer. Med., 1906, p. 105.

[10] Gesellschaft f. inn. Med., Wien, November 9, 1905.

[11] Compt, rend. Soc. de biol., 1903, p. 1659.

[12] Gazz. d. Osp., Milano, 1905.

[13] Wien. klin. Woch., 1905, Nr. 6.

The experimental production of these changes has introduced some other interesting problems, especially the question as to how these changes are produced and in what way they are related to arteriosclerosis in man.

Many theories have been advanced as to the direct cause of the process, as high tension, toxic influence, spasm of the vasovasorum, and cachexia. Fisher[16] believed the associated cachexia due to the prolonged use of the adrenalin was an important factor. Against this view is the rapidity of the process in many cases, also that no apparent cachexia can be detected in some animals in which marked calcification may be found. Erb considered spasm of the vasovasorum as an important factor. As the adventitia, as well as the media, receives its blood supply through this source, it is probable that both would be affected in case the changes originated in this way. A microscopic study has also failed to furnish any evidence directly supporting this way. The toxic and high tension theories have the strongest support at the present time.

In favor of the high-tension view is the sudden and marked rise in pressure following the injection of adrenalin. The strain thrown suddenly upon the vessel wall is very great, and might, as a result of trauma, lead to the changes in the media. It does not necessarily follow that, inasmuch as high pressure is a factor in the arteriosclerosis in man, it is necessarily associated with that in rabbits, since the two processes are usually different in character, affecting different coats in the vessel. Many, although not all, of the substances capable of producing these changes increase blood pressure. Phloridzin, with which Kolisch produced characteristic changes, does not increase pressure, the effect if any being to depress it. Bacterial toxins lower the pressure and lead per os has no demonstrable effect. Ludwig Braun thought he had demonstrated that increase in pressure is not the chief factor in adrenalin arteriosclerosis by attempting to inhibit the pressure effect through administering simultaneously amyl nitrite. He reports prompt and marked arterial changes in rabbits treated in this manner. If later experiments verified his results, we would have here the best of evidence that increase in pressure is at least not the only cause. Our experiments have shown that amyl nitrite even when given very freely does not inhibit the action of adrenalin. If the amyl nitrite precedes the adrenalin there is an initial fall but not a subsequent sharp rise; if they are injected simultaneously, the adrenalin acts so much more quickly that the rise takes place anyway; and it may be that the degree of change rather than the absolute height of pressure would produce arterial trauma. Sturli's[17] positive results with the subcutaneous use of adrenalin has also been advanced as evidence that the changes are toxic, for the reason that when given in this manner it does not increase pressure. In our experiment it has been demonstrated that 1 c.c. subcutaneously frequently causes a rise in pressure almost as great as when given intravenously, although the form of the curve is quite different. Meltzer and Auer[18] have shown also that in rare instances a moderate rise follows its subcutaneous use. In the light of these results, Sturli's proof is not convincing. Kulbs[19] and Josue after prolonged subcutaneous injections were unable to produce any arterial changes; however, the daily amount used was not very large. Intraperitoneally, adrenalin does not increase pressure, and Erb was unable to bring about any vessel changes after prolonged use in this manner. It might be mentioned before leaving this phase of the subject that Lissauer,[20] in order to prove that the changes are not due to high pressure alone, injected into rabbits 0.05 gm. per kilo of caffeine. This caused a rise in pressure of 60 mm., while adrenalin increased it 100 mm. He treated the animals for seven months, but was unable to produce vessel changes or heart hypertrophy.

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[14] Berl. klin. Woch., 1904, xxxi.

[15] Compt. rend. Soc. de biol., 1905, p. 219.

[16] Münch. med. Woch., January 3, 1905.

There is considerable evidence that extracts of the suprarenal have a toxic action quite independent of their vasoconstriction effect. Large doses administered subcutaneously cause death with symptoms of asphyxia without any decided increase in blood pressure. Elliott[21] and Tuckett discovered that the suprarenal gland of guinea-pig contains a substance which causes rapid necrosis of the guinea-pig's tissues. This is present in the cortical portion of the gland and is therefore not the pressor substance. Paton and Drummond[22] determined that the subcutaneous injection of adrenalin produces cloudy swelling and desquamation in the convoluted tubules of the kidney and central necrosis of the liver similar to that produced by certain bacterial toxins. Foa[23] several years ago reported degenerative changes, especially in the kidney, following intravenous and subcutaneous use of a watery extract of the gland. He reports that heating to 60° destroyed the substances causing tissue necrosis, but left the pressure substance intact.

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[17] Wien. med. Presse, 1905. S. 544.

[18] Jour. Exper. Med., vol. vii, No. 1.

[19] Archiv. f. exp. Path., Bd. liii.

[20] Berl. klin. Woch., 1905, 3675.

[21] Referred to by Elliott, Eng. Jour. Physiology, vol. xxxii, p. 464

[22] Eng. Jour. Physiology, vol. xxxi, p. 81.

[23] Archiv. Ital. de Biol., xxv, p. 250.

A review of the above evidence enables us to say: First, that arterial changes of this character may be formed independent of an increase in pressure; second, there is evidence that the extract of suprarenal has a toxic action. No satisfactory evidence can be found, however, that these changes are solely the result of either pressure effect or toxic action. If the result of pressure, it is probably due to the sudden rise rather than the prolonged effect—as after the administration of adrenalin, the pressure returns to normal within three to six minutes. Erb has suggested that there is a permanent increase in pressure following the prolonged use of adrenalin and has found a pressure of 160 mm. in a rabbit after long-continued injections.

The second question to be discussed is the relation of these changes to those occurring in man. Under the term arteriosclerosis we have several distinct pathological processes. In the usual form there is a hyperplasia of the intima, the media showing very slight if any changes. Calcification, if it occurs, is in the intima. Fränkel[24] has described another type in the peripheral vessels in multiple neuritis, tabes, and general paresis. He found quite uniformly marked hypertrophy of the media, the lumen being often almost obliterated. By cutting the sciatic nerve in rabbits he was able to produce similar changes in the vessels of the extremity. He considers angiospasm as the chief factor in this group of cases. The syphilitic aortitis, which is usually considered under the general term of arteriosclerosis, is a focal process confined to the media. However, it is inflammatory in character and shows little tendency to calcification, but rather to cicatricial contraction. Finally, we have the vascular changes described by Monckeberg,[25] who studied especially the peripheral vessels in elderly people. Fifty-five of the cases studied had focal degeneration of the media with calcification, the overlying intima somewhat thickened. When, *intra vitam*, the radial was hard and tortuous the chief change was a medial calcification. He concludes that medial calcification is more frequent in the peripheral vessels than thickening of the intima alone. Monckeberg's findings, so far as I have been able to determine, have never been refuted. Although he has been unable to study the early changes, as has been done in the experimental work on rabbits, it must be admitted that the similarity of the two conditions is striking.

My experiments were instituted in the hope of throwing some light upon the cause of vascular change: (1) By administering adrenalin with some substance that would inhibit its pressure effect (2) by giving it a through channel that might allow its toxic effect without its pressure action; (3) by attempting to destroy its toxic qualities and leaving the pressure substance intact; and (4) by producing experimental arterial degeneration with substances which have only a moderate pressure action.

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[24] Wien. klin. Woch, 1896, S. 147.

[25] Virchow's Archiv, Bd. clxxi, S. 141.

Brodie and Dixon[26] have reported that apocodeine, an alkaloid obtained from codeine by dehydration, when injected into rabbits or dogs causes a fall in pressure due to paralysis of the vasomotor nerve cells. The blood pressure can only be lowered to a certain fixed point beyond which further injection either has no effect or causes a slight rise in pressure. These large doses inhibit the pressor action of adrenalin. We gave rabbits 30 to 40 mg. subcutaneously and produced a marked fall in pressure, then administered the adrenalin, and, contrary to Dixon's results, had a prompt and decided rise. When larger doses of apocodeine were used the animals succumbed and our experiments with this drug were abandoned. Adrenalin when administered after chrysotoxin, the active principle of ergot, is said to cause a fall in blood pressure. Our experiments here also failed to produce the desired results, as there was always a prompt rise following the use of the adrenalin. Amyl nitrite, nitroglycerin, and sodium nitrite were next used. All of this group produce a marked fall in the pressure; however, when followed by adrenalin, the characteristic rise occurs, which we are unable to prevent even when very large amounts of the above substance were given. Three minims of the 1 to 1000 adrenalin chloride solution was used intravenously. The resulting absolute pressure is much lower than when these vasodilators are not given, but the degree of increase is practically unchanged. Although we are unable to inhibit the pressure action by this means, four rabbits were given this combined treatment. The absolute pressure being much less, negative results would favor the extremely high pressure as an important factor in the arterial changes. Amyl nitrite was selected as the most satisfactory of this group. At first, three minims of this was combined with an equal amount of the adrenalin and injected directly into the ear vein. As this set up a marked cellulitis, a mask was made and the rabbit inhaled the amyl nitrite while receiving the injection of adrenalin. Experimentally, it was found that by this method there would be a fall in pressure of about 40 mm.; when the amyl nitrite was inhaled, following the injection of the adrenalin, there would be the characteristic rise averaging about 80 mm., the blood pressure reaching on an average 140 mm., the normal pressure of the rabbit being about 100 mm. If amyl nitrite was not given, adrenalin would bring the pressure up to about 180 mm. By this means we were enabled to lessen the rise in pressure by about half without interfering with any possible toxic action of the adrenalin. The four rabbits received twenty-five injections of three minims each extending over a period of thirty-five days. At the end of this time they were chloroformed and the heart, thoracic and abdominal aorta examined. None of the vessels showed any gross or microscopic evidence of arterial degeneration nor was there any evidence of heart hypertrophy. These results do not agree with Braun's. His method of procedure, however, was different, as he mixed and injected both simultaneously. When this is done the action of the adrenalin, being more prompt than the amyl nitrite, an initial fall did not occur, as was the case when amyl nitrite was inhaled. These negative results with this small number of animals should not be looked upon as conclusive; it is, however, suggestive, as in my experimental work with adrenalin alone an average of at least half of the animals showed marked arterial changes. If in the light of the small number of animals used we should be allowed to draw any conclusions from this experiment it would be that high pressure is a factor in producing arterial changes.

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[26] Eng. Jour. Phys., vol. xxx, p. 476.

Following the suggestion of Foa, two rabbits were given intravenous injections of adrenalin chloride that had been previously heated to 70° C. This does not lessen its pressure action, but Foa thought it destroyed its toxic effect. We must bear in mind, however, that he used a watery extract of the gland and not the active principle. These animals received twenty-five injections of three minims each, over a period of five weeks. The autopsy showed slight changes in the thoracic aorta of one; very marked changes in the other. Thus it is seen that if the vascular change caused by adrenalin were due to a toxic substance it is not destroyed by heat.

Two animals were given daily subcutaneous injections of 1 c.c. of adrenalin for a period of five weeks. One animal became very much emaciated, due probably to a suppurative process in one eye and should have been a fit subject in which to produce arterial degeneration. However, in neither of them were there any vascular changes. These results are in accordance with the majority of those who have used adrenalin in this way. Inasmuch as a marked rise in pressure following the subcutaneous injection is quite inconstant, it is possible that Sturli's positive results might have been due to the use of rabbits that responded by a marked rise.

Two animals were given intravenous injections of 1.5 mg. of nicotine every day for six weeks. One of these showed slight but typical changes in the ascending aorta. Nicotine given in these doses intravenously causes first a slight fall in pressure, after a few seconds a sharp and marked rise, the character of the curve resembling that of adrenalin. Arterial changes due to its use may readily be considered a result of its pressure effect. It would be of interest to determine if tobacco given per os, as in Boveri's experiments, causes a rise in pressure; if negative, it would speak for the probable toxic character of the lesion.

At the suggestion of Dr. A. P. Matthews, of the University of Chicago, four animals were given intravenous injections of physostigmin. In doses of 1 mg. this causes a gradual and moderate rise in pressure, which returns to normal in six to eight minutes. The action is a direct muscular one, affecting both the heart and vessels. The animals received daily 1 mg. for a period of eight weeks. Two of these showed a single focus of degeneration in the descending aorta. The gross appearance differs slightly from that of adrenalin. There was a distinct, single, mound-like elevation of the wall; in one the calcareous deposits could be readily detected by the finger; in the other there was merely a sense of greater resistance than in the surrounding wall. Microscopically the changes have not yet been carefully studied.

Very interesting results were obtained from the use of barium chloride. When 1 mg. is injected intravenously into a rabbit there is a gradual and moderate rise in pressure of 25 to 35 mm. This rise is maintained for a half hour or more, and is the result of a combined action upon the heart and bloodvessels. Compared to adrenalin the rise is more gradual, much less in degree, and much more prolonged. Three rabbits were given 1 mg. of barium chloride intravenously. One of them received twenty-two injections within fifty-six days; the others twenty-two injections each, extending over thirty-five days. The animal treated for eight weeks showed a single mound-shaped elevation, the size of a pinhead, in the transverse aorta. Macroscopically there was no evidence of calcification. One of the other rabbits did not show anything abnormal; the other, in the aorta just above the valves, had numerous small elevations. These varied in size from a small pinhead to two or three times this diameter. They were soft to the touch. Under a hand-glass they appeared as mound-like elevations irregular in outline and furrowed. Over these areas the glistening appearance of the intima was lost, being replaced by a dull gelatinous appearance. The picture differed from any that I have seen following the use of adrenalin, where from the onset the process is a destructive one, no thickening of the wall being observed. The microscopic changes differ quite distinctly from those previously described. The changes appear to be confined chiefly to the subendothelial layer of the intima. The differentiation between intima and media is not sharply defined in the rabbit's aorta, as the amount of muscle tissue in the latter is slight. It was the opinion, however, of Dr. Bensley, of the Chicago University, who kindly examined my specimens, that the changes to be described are in the intima rather than the media. This is also the impression gained from the examination under low magnification, as the area of involvement appears as a distinct narrow band paralleling the endothelium. The fine wavy outline of the elastic fibers in the involved areas is replaced by a marked tortuosity.

The interspaces between the fibers are markedly increased and it is this apparently that leads to the thickening of the wall. The fibers in the adjacent media are often compressed as are also often some of the overlying fibers of the intima. The fibers were furthermore irregularly swollen and altered in their staining qualities. With iron-hematoxylin they appear intensely black, with the toluidin-blue sections of the fibers stain giving a beaded appearance. Where calcification occurs the lime salts are deposited between the fibers. These changes require further study, which I hope soon to carry out on another series of rabbits. Of interest, however, is the demonstration of changes in the elastic fibers and the possibility of the process being chiefly confined to the intima. Certainly, the changes in many respects differ from those following the use of adrenalin. No noticeable heart hypertrophy was observed in these animals.

In conclusion we can say there are a number of substances capable of producing vascular changes. At the present time this list includes bacterial toxins, extract of suprarenal and various similar synthetical preparations, phloridzin, digitalis, nicotine, physostigmin, barium chloride, and lead. The majority, but not all of these, increase the pressure. This may be largely accounted for by the fact that those who have done experimental work have selected those substances that increase pressure, as they have considered this an important factor in the change. More work should be done with other groups of substances, and then it may be demonstrated that increased pressure is not essential for the changes.

Our work has added two new substances to the list of those capable of producing arterial changes—physostigmin and barium chloride. Both of these cause an arterial degeneration, the latter producing changes apparently differing from adrenalin. We have demonstrated that the subcutaneous injection of adrenalin may cause a very distinct rise in pressure, thus annulling the evidence, that when arterial degeneration follows the subcutaneous use of this drug, it is necessarily due to its toxic rather than its pressure effect. We were also unable to inhibit the pressure action of the adrenalin by the use of amyl nitrite. For this reason arterial changes following the combined use of these substances does not prove that the process is toxic, as was maintained by Braun.

I wish to express my gratitude to Drs. S. A. Matthews, and A. P. Matthews of the department of physiology, and also the various members of the department of anatomy of the University of Chicago, for their very valuable assistance.

SOME CONGENITAL ANOMALIES OF THE HANDS AND FEET.

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The accompanying plates illustrate some congenital anomalies which came to our notice at the Presbyterian Hospital in New York during the last year. For the excellent photographs I am indebted to Mr. Ira L. Simms, of the *x*-ray Laboratory of the Hospital.

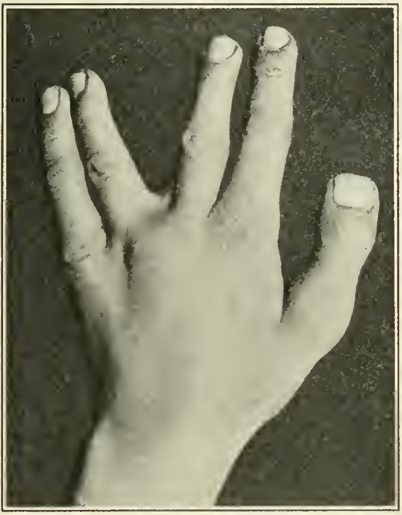
Case I.—The ordinary photographs of the hands show prominence of the third and fourth metacarpal bones, which in the *x*-ray photographs are seen to be fused together. The ring fingers articulate at an angle with these consolidated bones. Note also the breadth of the last phalanges of the thumbs, where there were evidently attempts made to form two distinct distal phalanges in each thumb. This patient was a mechanic and suffered no disability.

Case II.—Illustrates a condition of webbing of the middle and ring fingers of each hand. In the left hand the bones of an extra digit are well-formed, though they are small. In the right hand this extra digit has not become separated from the ring finger. There is also absence of a joint between the first and the second phalanges of the right hand.

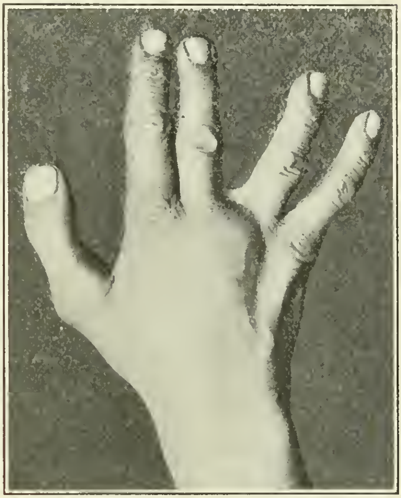
Case III.—The two plates represent the hands and feet of the same patient, each of which has six digits, except the right hand, which is normal. The extra digit on the left hand is formed by a branching of the innermost metacarpal bone at its distal extremity. The extra digit which has three phalanges is an extra ring finger. Each foot of this patient has an extra digit on the outer side of the foot, each formed by a branching of the fifth metatarsal bone. Note the difference in the manner of this branching on the two sides.

Case IV.—Supernumerary left thumb. The last two phalanges are ankylosed at the distal joint and arise from a branched metacarpal bone; note also the partial dislocation of the supernumerary portion on the metacarpal. The extra thumb is practically useless and in the way; the right thumb is normal.

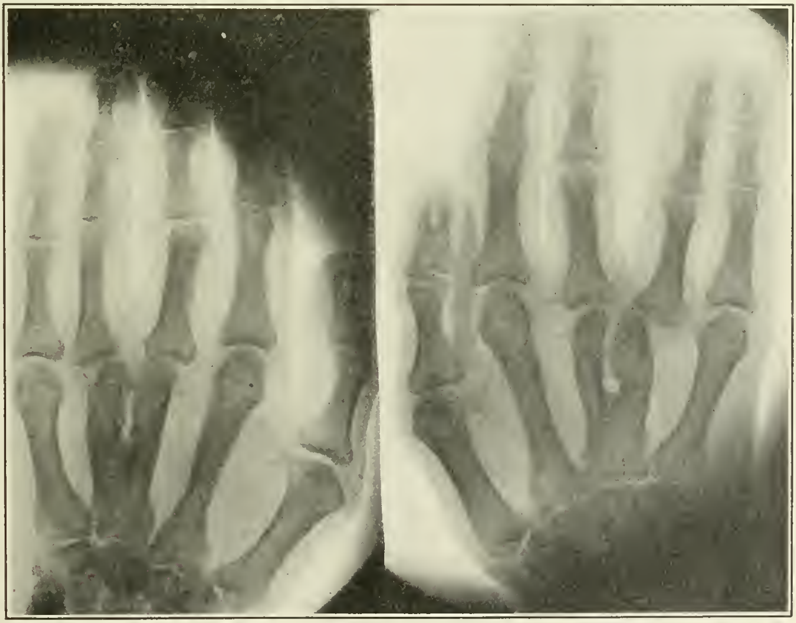
Case V.—Represents congenital absence of one forearm and three fingers in a boy six years of age. The articular surface on lower end of the humerus is practically absent as are entirely the ulna and the radius. Below the humerus there is a carpal bone. Other carpal cartilages are probably present (*x*-rays do not show cartilage), and will ossify later, but at the present time only one carpal cartilage is ossified. The hand is represented by two fingers, which can be used by the boy to an astonishing extent in most useful ways. Motions at the wrist and in the fingers are good. One of the fingers is evidently a thumb, for the following reason: by comparing the normal metacarpal



CASE I.—Left hand.

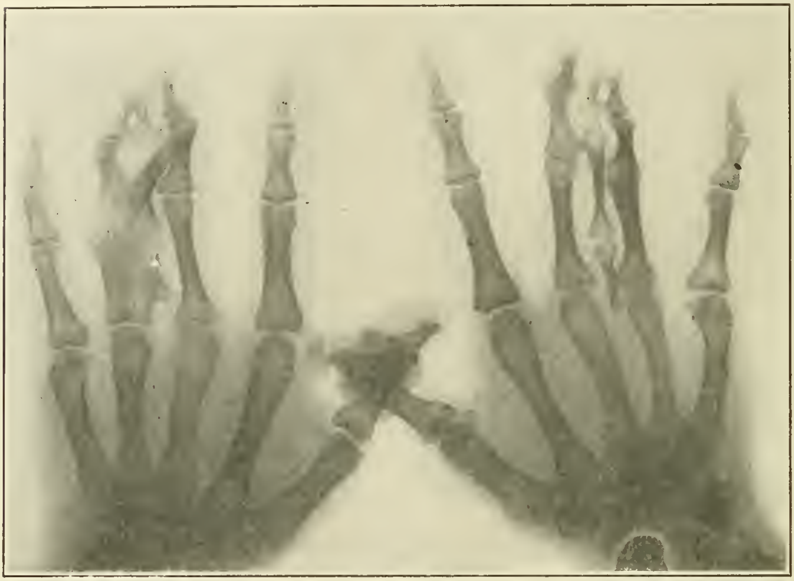


CASE I.—Right hand.

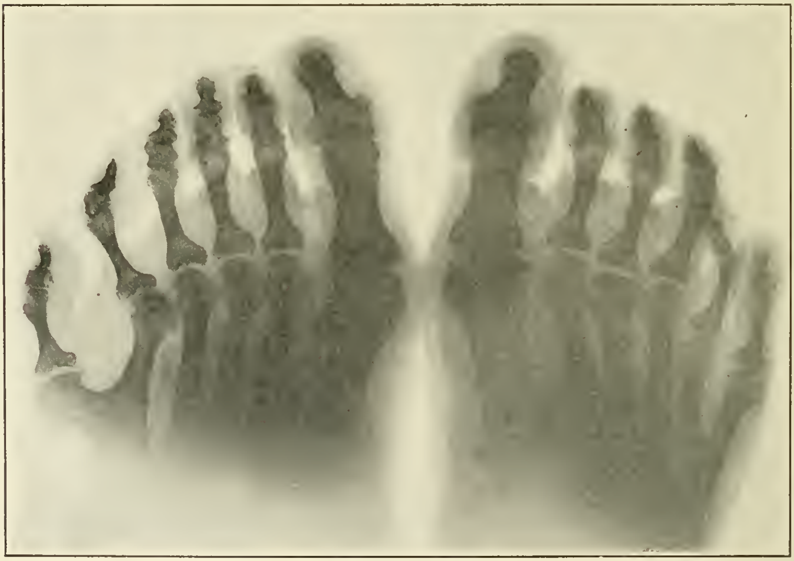


Left hand. CASE I. Right hand.

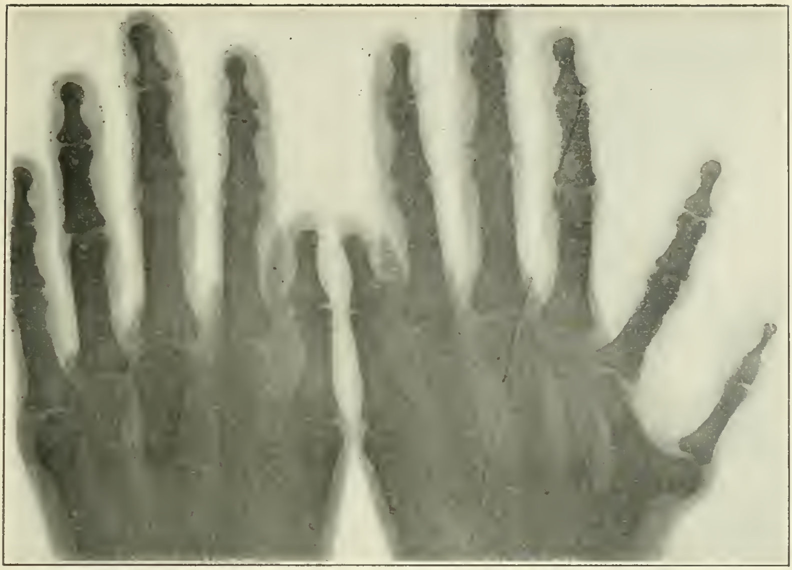
bones of the hands of a boy of ten in Case VI, we see that the normal epiphyseal line in the metacarpal bone of the thumb is situated on the wrist end of the metacarpal, while in the metacarpals of all the fingers the epiphyseal lines are situated on the phalangeal ends.



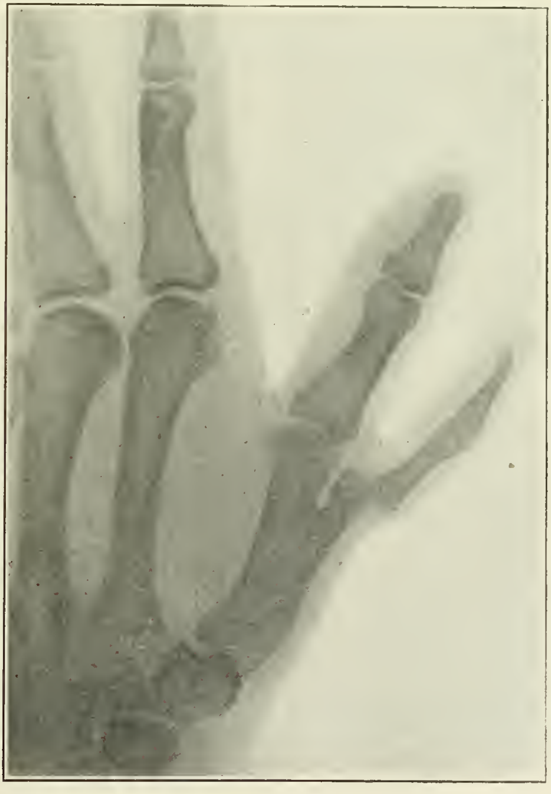
Right hand. Case II. Left hand.



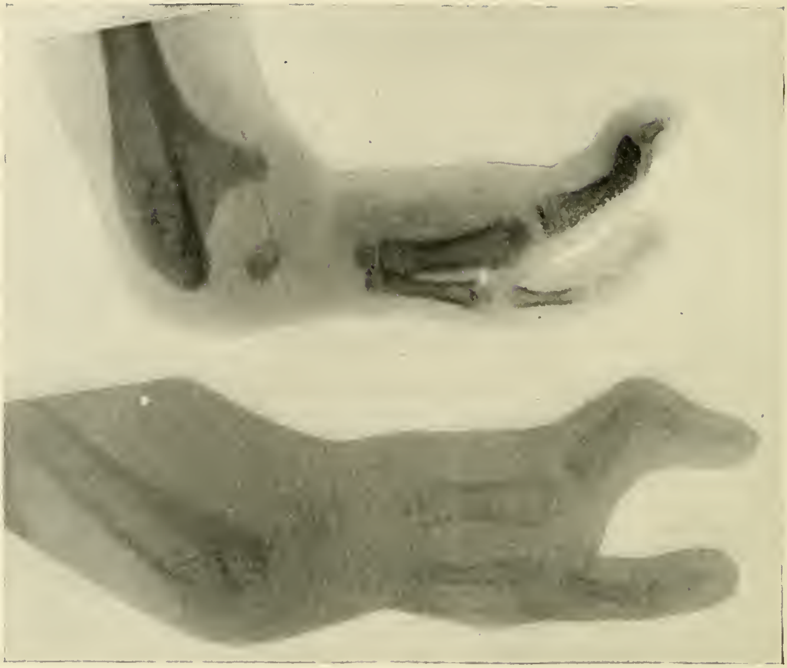
Case III.—Feet.



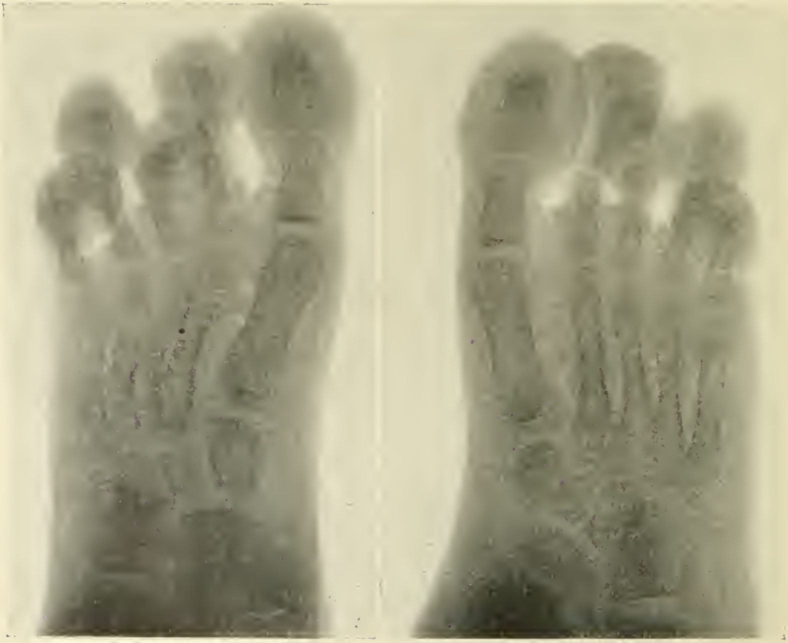
Right hand. CASE III. Left hand.



CASE IV.

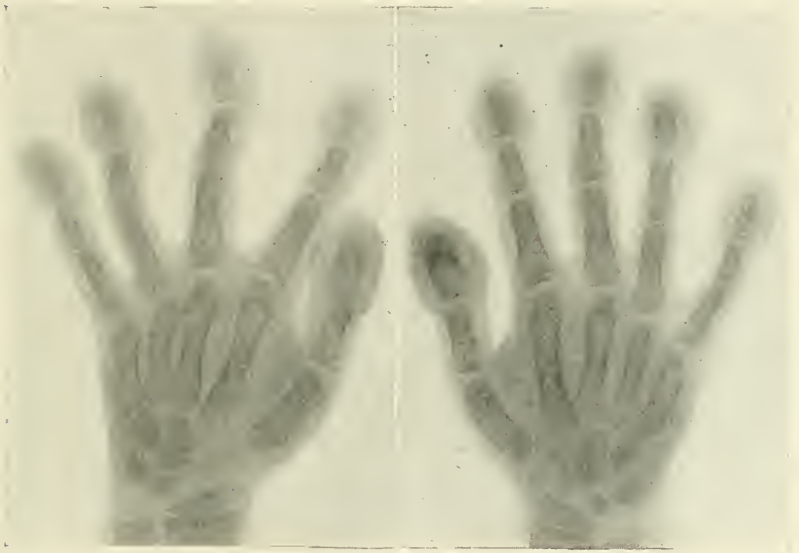


Case V.



Case VI.

In Case V we see that in one metacarpal the epiphyseal line is on the phalangeal end, so that we may say this is a true finger while the other is a thumb, because its epiphyseal line is on the wrist end of its metacarpal. Note also the unusual fact of there being two epiphyseal lines on the thumb metacarpal. The *x*-ray photographs of the two fingers were taken at different angles. The opposite forearm and hand are normal.



CASE VI.

CASE VI.—Hands and feet of a boy of ten, illustrating clubbing of the fingers and toes. The boy had fibrosis of one entire lung consecutive to an unresolved bronchopneumonia; nephritis also was present to a marked degree. Notice the feathery thickenings of the distal portions of the last phalangeal bones. The clubbing consists not only of thickening of the soft parts but also of the bones of the last phalanges, due to a productive periostitis.

THE STANDARDS AND WORK OF THE PHILADELPHIA PEDIATRIC SOCIETY'S MILK COMMISSION.

EMBODYING A REPLY TO A CRITICISM BY DR. A. H. STEWART, OF THE PHILADELPHIA BUREAU OF HEALTH.[1]

By Samuel McC. Hamill, M.D.,

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The Milk Commission of the Philadelphia Pediatric Society was appointed in January, 1899. A close relationship between the milk supply and the high infant mortality, especially in the summer months, had been recognized for many years. Many efforts had been made to improve the conditions governing milk production. Pasteurization and sterilization had been resorted to, the various proprietory foods had been extensively used, and all without any material influence upon the death rate.

It was thought by the Pediatric Society that something might be accomplished in this line by interesting some of the modern well-equipped dairies in the production of a milk of a high degree of cleanliness, with a low bacterial content, and constant chemical make-up. With this end in view, a Committee was appointed to investigate the dairies of this class supplying milk to Philadelphia, and to consult with milk producers and veterinarians, as to what should constitute the standards under which a Milk Commission should work.

The report of this Committee, made to the Society in January 1900, embodied the following standards:

Bacteriological. The bacteriologist shall procure a specimen of milk from the dairy, or, preferably, from delivery wagons, at intervals to be arranged by the Commission and the dairy, but in no case at a longer interval than one month. The exact time of the procuring shall be without previous notice to the dairy. He shall test this milk for the number and nature of the bacteria present in it, to the extent which the needs of safe milk demand. He shall also make a microscopic examination of the milk for pus cells. Milk free from pus and injurious germs and having not more than 10,000 germs of any kind or kinds to the cubic centimeter shall be considered up to the required standard of purity.

Chemical. The chemist shall, in similar manner, procure and examine the milk for the percentages of proteids, fat, sugar, mineral matter, and water present. He shall also test its chemical reaction and specific gravity, and shall examine it for the presence of foreign or other matters, or of chemicals added as preservatives. Standard milk shall range from 1029 to 1034 specific gravity, be neutral or very faintly acid in reaction, contain not less than from 3.5 per cent. to 4.5 per cent. proteid, and from 4 per cent. to 5 per cent. sugar, and not less than 3.5 per cent. to 4.5 per cent. fat, and shall be free from all contaminating matter and from all addition of chemical substances or coloring matters. Richness of cream in fat shall be specified, and shall vary not more than 1 per cent. above or below the figure named in selling. Neither milk nor cream shall have been subjected to heat before the examination has been made, nor at any time unless so announced to the consumer.

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[1] Amer. Jour. Med. Sci., 1906, cxxxi, 625.

VETERINARIAN. The Veterinary Inspector shall, at intervals equal to those of the bacteriologist and chemist, and without previous warning to the dairy, inspect the cleanliness of the dairy in general, the care and cleanliness observed in milking, the care of the various utensils employed, the nature and quality of the food used, and all other matters of a hygienic nature bearing upon the health of the cows and the cleanliness of the milk, including also, as far as possible, the inquiry into the health of the employés on their farms. He shall also see that the cows are free from tuberculosis or other disease.

DUTIES OF EXPERTS. The experts shall make their examinations when, and only when, notified to do so by the Commission. Any dairy, the milk of which shall be found by the examiners to be up to the standard of the Commission, shall receive certificates from the Commission.

In case an examination shows the milk not up to the standard, the dairy may have a re-examination made within a week, or within a short time, at the discretion of the Commission.

Milk furnished by the producers to whom certificates have been issued shall be delivered to consumers in glass bottles hermetically sealed in a manner satisfactory to the Commission. In addition to the sealing, and as a guarantee to the consumer that the examination has been regularly conducted, there shall be pasted over the mouth of the jar, or handed to the consumer with every jar, according to the discretion of the Commission, a certificate slip which shall read as follows:

CERTIFICATE OF THE PHILADELPHIA PEDIATRIC SOCIETY'S MILK COMMISSION.

Milk from the dairy of Mr. .... has been recently examined by the experts of the Milk Commission and found to be fully up to the required standards. Another examination is to be made within a month, and, if satisfactory, new labels for the bottles will be issued, dated..................

Notice the dates.

This report was approved by the Society and was adopted as the standard of the Commission. Since its adoption it has been changed in the following particulars:

1. The method originally adopted, for the collection of the samples for examination by the experts, was open to the following objections: It was impossible for either of them to obtain samples from the delivery wagons without previously notifying them to call at their laboratories, and it was equally impossible to secure reliable messengers to carry the milk to them from the delivery stations. At the suggestion of Prof. R. A. Pearson, of Cornell University, then General Manager of the Walker-Gordon Company, it was decided to require the producer, or his agent, to make daily deliveries of samples of milk to the house of the Secretary, thus enabling the Commission to select samples for examination at its discretion. Otherwise the bacteriological requirements remain the same, save that the Commission now certifies to cream, the conditions governing its certification being the same as those for milk, except that it is allowed a bacterial content of 25,000 to the cubic centimeter.

2. The chemist, instead of receiving samples direct from the distributer, examines the samples previously studied by the bacteriologist. The specific gravity of the milk is no longer determined, the acidity of the milk is definitely determined, the allowed variation of the proteid content has been changed to from 3 per cent. to 4 per cent., and the variation in the fat content of the creams has been permitted to vary 2 per cent. on either side of the percentage specified on the bottles. The Commission now certifies to 5 per cent. fat milks, allowing the same variation as in the case of the 4 per cent. milks.

3. The certificates issued to the producers are now placed between the pulp, and tinfoil or paper caps, covering the top of the jar.

These standards, in the experience of the Commission, have proved entirely satisfactory. Some of the dairies which have been under the certification of the Commission since its organization were unable to maintain these standards. These have either been dropped or withdrawn from our certification. Some others, whilst never producing a milk of high bacterial content, have had some difficulty in meeting our requirements, and all have failed occasionally.

The chemical and veterinary requirements have been met more satisfactorily than the bacteriological. There have been practically no failures in the proteid content since the variation of from 3 per cent. to 4 per cent. was determined upon. One of the dairies, producing a 4 per cent. fat milk, had some difficulty for a few months in keeping its milk within the required fat limits. By intelligent shifting of the herd and the introduction of new cows, this obstacle has been definitely overcome.

The Commission feels that it has accomplished even more than it had in mind at the time of its organization. It has not only put at the disposal of the Philadelphia public, milks and creams of a high degree of purity, probably higher than that of any milks and creams produced in the world, but it has stimulated an interest in the pure milk question throughout the country. What is probably of more importance, certainly to Philadelphians, is the fact that it has stimulated the producers and dealers to take greater precautions in the preparation and distribution of milk, thereby elevating the standard of the general milk supply.

There has been an occasional criticism of the rigidity of the standards required by the Commission, and there has recently appeared an article by Dr. A. H. Stewart,[2] which the Commission considers unjust. The article in question is based upon the results of a long series of bacteriological examinations of the various milks receiving the certification of the Commission, together with one milk certified to by the Company producing it and one other which withdraw from the certification of the Commission during the course of these examinations.

Dr. Stewart's article would have been of more practical and scientific value had it contained a definite description of the methods pursued in reaching his results; the manner of production of the media; the temperature at which the plates were incubated; the length of time of the incubation, etc.; a statement of the principle upon which he determines what number of leukocytes in the milk constitutes pus, and especially the exact method by which the inspectors of the Bureau of Health received and conveyed to the laboratories the samples which were examined.

In making his bacterial counts, Dr. Stewart presumably, although he does not so state, made one examination immediately upon the receipt of the samples and another at the end of twenty-four hours. The second examination does not concern the Commission, inasmuch as the keeping of the milk after its delivery is a responsibility which must be assumed by the consumer and is, therefore, beyond the jurisdiction of the Commission. The only comment we need make thereon is that the results obtained would seem to reflect upon the manner in which Dr. Stewart kept the milks which he examined.

As to the manner of collecting the milk we would say that, unless the milk was placed on ice and kept on ice from the time it was accepted from the dealer until the time of the examination, the results of the bacteriological counts would prove unjust to both the producer and dealer. It is the custom of the Commission to have all samples delivered to the house of the Secretary under exactly the same conditions governing the delivery to the ordinary consumer. When the milk is so delivered the responsibility of the producer and the dealer ceases. Recognizing this, all samples are immediately placed upon and kept on ice until the bacteriologist's examination is made, in order to hold the bacterial content, as nearly as possible, the same as at the time of delivery.

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[2] Amer. Jour. Med. Sci., 1906. cxxxi, 625.

Let us now consider the results of Dr. Stewart's bacteriological examinations:

In the case of Dairy No. I (following Dr. Stewart's system of numbering) he made fifty-four examinations between July 18, 1904, and August 24, 1905. Of this number, he condemned 31 (57.4 per cent.), seven for containing pus cells—which means that he considered all samples to contain pus showing a leukocyte count above 100,000 per cubic centimeter; seven for containing streptococci; and twenty-nine for containing bacteria in excess of 10,000 per cubic centimeter—the standard required by the Commission. The samples showing bacteria present in excess of 10,000 per cubic centimeter gave the following counts: 10,200; 10,500; 11,000; 11,300; 11,700; 12,320; 14,600 twice; 16,200; 16,300; 16,500; 18,000; 27,000; 49,700; 72,000; 86,260; 93,000; 94,000; 102,400; 113,000; 117,000; 120,000; 132,750; 150,000; 200,000; 279,000; 390,000; 500,000; 770,000; in other words eleven of these twenty-nine samples contained less than 20,000 bacteria per cubic centimeter and eleven samples over 100,000 per cubic centimeter. In the experience of the Commission, as well as in the results of Dr. Stewart's work, this dairy has made the poorest showing of any of the dairies under the certification of the Commission.

Dairy No II, in Dr. Stewart's series, is the one which has not been under the certification of the Commission since September, 1904, and will therefore not be considered.

Dairy No III had fifty-four samples examined, of which two were condemned for pus cells—(140,000 and 104,000 leukocytes), one for streptococci, and, according to Dr. Stewart's statements, nine times for a bacterial content in excess of 10,000. In his column of results, however, there are but seven counts above 10,000. There is one of 10,000 and the seven others are as follows: 14,000; 15,670; 30,000; 37,500; 50,000; 78,000; 80,500; none above 100,000 and but two above 50,000.

The results of the examination of milk from Dairy No. IV, which Dr. Stewart wrongly states received the certificate of the Commission until June, 1905—the last issuance of certificates to this company having been in January, 1905—will not be considered after the date covered by the last certificates granted it, namely, up to February 10, 1905. This leaves twenty-eight examinations for consideration. In this number, streptococci were found four times, more than 100,000 leukocytes once, and bacteria in excess of 10,000 to the cubic centimeter eight times. Of this number, the counts were 11,250; 14,500; 15,500; 21,130; 24,000; 24,650; 27,400; 730,000. The count on two occasions only was above 25,000 and once only above 100,000.

Fifty-two examinations were made of the milk from Dairy No. V. Of this number, four were condemned for pus cells, none for streptococci, and ten for a bacterial content in excess of 10,000 per cubic centimeter. In these instances the counts were as follows: 10,938; 11,200; 11,600; 12,000; 13,000; 14,800; 28,000; 29,300; 44,000; 77,000. In other words in only four instances was the bacterial content above 25,000 and but once above 50,000.

Fifty-two samples were examined from Dairy No. VI. Pus cells were never found; streptococci were present three times, and bacteria in excess of 10,000 per cubic centimeter nine times. Of this number the counts were 11,000; 11,500; 12,460; 14,300 (twice); 14,400; 15,800; 20,000; 60,000. In only one instance, therefore, was the number of 20,000 exceeded, and that count showed but 60,000 per cubic centimeter.

To sum up the results of these investigations, we find, excluding the results in Dairy No. I, that in 186 examinations the milk contained streptococci five times and exceeded a bacterial content of 10,000—thirty-four times, or in 18.28 per cent. In twenty-two instances the bacteria present numbered 25,000 or less; but three times more than 50,000, and but once more than 100,000; results which must be considered remarkable. Including Dairy I, which was formerly producing the poorest milk of any we certified to, but which is now doing excellent work, the number of examinations made was 240. In this number streptococci were present twelve times and the bacterial content of 10,000 was exceeded sixty-three times or in 16.25 per cent. In thirty-four instances the bacterial count was 25,000 or less, more than 50,000—eighteen times, and above 100,000—twelve times.

It should be observed that the twenty-nine failures of Dairy No. I to meet the bacteriological standard almost equals the combined failures—thirty-four—of the four other dairies, and that the number of instances in which streptococci were found—seven—exceeded by two the number in all of the other dairies. At the time of these examinations, the output of Dairy No. I amounted to less than one-third the total output of all the dairies. Any criticism, therefore, which is based upon the total results obtained from an examination of all the dairies is decidedly unfair to the four dairies making such excellent showings as well as to the Milk Commission.

In reference to what constitutes pus in milk, the views of the bacteriologist of the Milk Commission are not in accord with those of Dr. Stewart. The opinions which Dr. Ravenel holds in regard to this matter have been kindly written for me by him, and they are as follows:

"PUS IN MILK. In taking up the work for the Milk Commission, the bacteriologist realized at once that the question of pus in milk was a most difficult one and the matter was laid before the Commission for discussion, and correspondence entered into with a number of authorities on milk. Leukocytes are practically always found in milk—so much so that we should consider them a normal content, or else hold the anomalous view that all milk comes from udders which are inflamed to a greater or less extent. The pus cell is identical with the leukocyte, and is in fact the leukocyte after leaving the blood. At what point then must we consider leukocytes in the milk as an evidence of disease and call them pus? No standard has been fixed and those who have tried to do so have reached widely varying results, and the methods employed have, with a single exception (Doane), been wofully lacking in scientific accuracy. Dr. Stewart fixes arbitrarily 100,000 per cubic centimeter as an indication of pus, and has condemned many samples of milk on this standard. The normal leukocyte content of the blood in man is about 1,000,000. It is perfectly conceivable that the blood may contain 1,000,000 leukocytes and be healthy, while 100,000 in milk, or some other secretion, may mean pus and inflammation, but to assume this without further proofs than have been brought forward is unscientific, arbitrary, and unjust to the milk producer. It has not been shown by anyone that milk high in leukocytes has any injurious effect, nor has it been shown that their presence means *per se* inflammatory changes in the udder. Certain foods are said to produce a leukocytosis in milk. Induration of the udder can be produced by the injection of sterile water. Leukocytes may be had in enormous numbers in certain fluids by the introduction of sterile substances, as for example aleuronat. It seems perfectly possible that during the process of milking, leukocytes may be squeezed out, so to speak, from the lymph spaces into the milk. It has seemed to the bacteriologist, therefore, that a high lenkocyte content should not of itself condemn a milk as containing pus. When it is found in a sample, plates made from the sample are carefully examined for the bacteria generally known to produce inflammation, such as streptococci and staphylococci. The presence of fibrin is also a most useful aid, as pointed out by Doane."

As has been previously remarked, Dr. Stewart does not state by what process he determines the presence of pus in milk, but, in the light of his figures, as Dr. Ravenel has said, one must assume that any milk containing more than 100,000 leukocytes to the cubic centimeter is a pus-containing milk. A number of attempts have been made to establish a standard by which it could be determined what number of leukocytes constitute pus. The earlier methods adopted for this purpose have been shown to be unreliable and up to the present time no standard has been reached.

The most recent, reliable, and comprehensive study that has been made on this subject emanates from the Maryland Agricultural Experimental Station and is presented by Charles F. Doane, referred to by Dr. Ravenel. This author does not consider it desirable to determine the presence of inflammation in the udder by the mere presence of a large number of leukocytes. We quote from his article in order not to misrepresent him and for purposes of clearness, as follows: "The presence of fibrin in pus gives rise to a fairly satisfactory and rapid means of determining the presence of pus in milk. The milk threads catch and retain a large number of leukocytes in a mass. . . . If no fibrin is present, the leukocytes will be distributed singly or at most five or six gathered together. If any quantity of fibrin is present, large numbers of leukocytes, from twenty to one hundred in a single mass, will be seen under the microscope. . . . There can be little doubt that these leukocyte masses or clumps furnish the most practical and easiest means of determining the presence of pus and the healthfulness of milk." As to the number of leukocytes to be found when other conditions suggest the presence of pus, he says: "With these additional means of distinguishing pus, it is interesting to know about how many leukocytes are actually present when milk evidently contains pus. . . . Judging from these and other figures we have of a similar nature, it would appear that milk with a leukocyte count of 500,000 per cubic centimeter is suspicious, when it reaches 1,000,000 per cubic centimeter there is undoubtedly inflammation in the udder."

Therefore, in the light of our lack of knowledge of what should constitute a standard for pus, and in the presence of this statement of Doane's, it seems eminently unfair that anyone should publicly condemn the milk of any dairy for containing pus on the presence of 100,000 leukocytes per cubic centimeter. It is really incomprehensible that one can determine that a milk containing 96,800 leukocytes is free from pus and that a milk containing 100,000 leukocytes is a pus-containing milk.[3] It is interesting to note that in the 297 examinations for pus made by Stewart, in only one instance would any of these milks have been considered to have contained pus on the basis of Doane's figures.

The criticisms which Dr. Stewart makes in concluding his article are as follows: (1) One examination of milk per month is not sufficient; (2) examinations should be made more frequently in the summer than in the winter; (3) examinations for pus and streptococci should be made before the milk is sold; and (4) extra expenses connected with the examinations should be abolished at once.

Answering these in the order in which they were written, I would say that statement No. 1 is incorrect. The minimum number of examinations required by the Commission is one in each month. When there are indications that the working of the dairy or the condition of its products are unsatisfactory, additional examinations are made. The Commission fully recognizes the fact that more frequent examinations would be much more satisfactory to itself as well as to the consumer, but its purpose in making the examinations which may be held at any time during the month, as few as possible, is to reduce the expense to the producer. The only entirely satisfactory method would require a daily examination of the milk, and the expense of such examinations, which must be borne by someone, would scarcely be consented to by the producer and could not be borne by the Commission or the Society which it represents.

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[3] See Stewart's figures. Amer. Jour. Med. Sci., loc. cit.

On the basis of the results obtained by Dr. Stewart, it is questionable if much more could be accomplished by having the examinations made with the same frequency as that adopted by him. Whilst he unquestionably detects failures more frequently than does the Commission, there are a number of instances in which with his frequent examinations he fails to note failures that are recognized by the Commission. The question is one which must be looked at from a practical standpoint and not from the theoretical viewpoint of a laboratory worker. It is an easy matter to sit in a laboratory and make a long series of examinations and evolve a theory therefrom that, if put into operation, might result in the elimination of bacteria in milk. Unfortunately, such theories are of no value. Whatever scheme is adopted for the improvement of the milk supply must take into consideration all of the difficulties under which the producer and dealer work, as well as the expense of the examinations. The purpose of any examination or any standard is to stimulate the producer to adopt the most modern equipment calculated to eliminate the objectionable features of milk production, and to establish a technique in its application as nearly perfect in all its details as is possible and to keep him constantly on the alert in carrying out the minute details of such technique. With the fact constantly before him that he has a definite standard by which his work is measured and that the test of his efficiency (the examination) is liable to be made on any day of the month, is, in the judgment of this Commission, a necessary and sufficient stimulus to good work.

The standard of 10,000 bacteria per cubic centimeter which is only a small part of the standard by which the Commission measures the work of the diary, we do not think, in the light of Dr. Stewart's results or those of our bacteriologist, too high. It is not expected by the Commission that this standard can always be maintained, nor can any useful standard which may be adopted, but we do know that during the most of the time it is, and that the effort on the part of the various producers to reach such a standard, results in the nearly constant production of a good safe milk—a milk so far above the ordinary market milks in point of cleanliness, constancy of its chemical constituents and freedom from pathogenic and all other forms of bacteria, that comparisons are impossible.

As to the necessity of more frequent examinations during the summer than during the winter months, the Commission is at variance with Dr. Stewart—a difference probably explained by the fact that Dr. Stewart made his examinations during two summers and one winter. In the experience of the Commission and in fact on the basis of Dr. Stewart's own figures, the percentage of failures is greater during the months of February, March, and April than during those of June, July, and August, a fact which is probably attributable to the unpreparedness of the dairies for the sudden elevations of temperature which occur at these seasons and to the prevalence of high winds.

Even during these months the Commission does not see that any advantage is to be obtained by increasing the frequency of the examinations, because the examinations which are made detect the failures, and when failures are noted these dairies or their products are kept under supervision long enough to assure the Commission that proper precautions are being adopted to meet the conditions producing the unsatisfactory results.

The recommendation that milk should be examined for pus and streptococci before it is sold is so absurd that it scarcely deserves comment. What does such a proposition involve? In the first place, the milk from an entire herd is never all mixed together. The milk from certain sections of the herd only is mixed, and, therefore, in the product of each dairy there are a number of mixed milks.

If these different mixtures could be kept separate, it would be necessary to have samples from each examined, and even if one sample represented the milk from the entire herd, the inconvenience to the dealer and the expense of employing someone to make such examinations would be too great to place the scheme within the range of possibilities.

The only objection to Dr. Stewart's suggestion that "extra expenses connected with the examinations should be abolished at once," is that he does not suggest the means by which this can be accomplished. It has been the constant purpose of the Commission to reduce the expense to the producer to the lowest point possible, and any suggestion that tends to accomplish this will be gladly welcomed.

In order that the work of its experts—the veterinarian, the bacteriologist, and the chemist—might be of the utmost value, the Commission selected the most eminent men working in these lines to do this work. It is done at the present time at great inconvenience to all of them and at a figure that absolutely fails to compensate them for their services. They happen, all of them, to be men who are deeply interested in the purposes of this Commission, a fact which enabled us to secure their services, and the work that they have done has been most painstaking, conscientious, and valuable. To them more than to any other persons, the Commission owes the measure of success which it has met, and it desires to take this opportunity publicly to express to them its appreciation of their services.

The statement made by Dr. Stewart that "at present there is no one to whom the dairymen can go for either instruction or advice concerning methods, and when trouble comes and the milk is high in bacterial count, he does not know where to locate the trouble," is incorrect. The duty and the practice of the veterinarian is to point out to the producer any deficiencies in his technique and to recommend measures for their correction. Any of the producers will testify to this. In fact one of the dairies and the Commission parted company because the owner objected to the criticisms and recommendations of the Veterinarian. The Commission has always aimed to be helpful to the producers—a statement which we believe will be endorsed by any of those to whose products we certify.

Dr. Stewart is also in error in his statement that most of the certified milk is "stored for twenty-four hours before it is delivered" and that the milk "delivered at the doors in the early morning hours" is usually two days old. None of the milks are stored for twenty-four hours and only part of the product of one dairy is forty-eight hours old when it reaches the consumer, but inasmuch as the product of this dairy—Dairy No III—makes the best showing bacteriologically of any of the milks which Dr. Stewart examined, this fact does not seem to have materially affected its quality.

The truth regarding the deliveries of the various dairies is this: The milk from Dairy I is twenty-four to thirty-six hours old when it reaches the consumer—not two days old, as Dr. Stewart says; the milk from Dairy III is thirty-six to forty-eight hours old; the milk from Dairy V is twenty-four to thirty-six hours old; and that from Dairy VI, twelve to twenty-four hours old. The Commission does not wish to be understood as commending the lateness of these deliveries and refers to the matter solely to correct Dr. Stewart's misstatements, which are unjust to the producer and dealer. The lateness of these deliveries is unfortunately necessitated by inadequate railroad facilities, a condition which the dealers of Philadelphia, as well as the shippers engaged exclusively in the dairy business, have endeavored unsuccessfully to overcome.

We accept with deep feelings of gratitude Dr. Stewart's statement that the Commission's "supervision of dairies has had a most excellent influence, an influence that is being felt in the general milk supply" and in this we believe he is correct, but again we cannot endorse his statement that "the bacterial counts of certified milk are rather disappointing." Our reports are better than his, but we are willing to submit his own figures in evidence.

In closing, the Commission wishes me to add a word regarding the pasteurized milk to which it certifies. At the time the Commission was organized little was known of the bacterial content of pasteurized milks. Pasteurized milks were considered safer than raw milks. The Commission assumed that if the milk which it examined in the raw state was safe from a bacteriological standpoint, it would be safe after pasteurization, and, therefore, decided to certify to it without examination, in order to reduce the cost to the producer. In the light of the examinations of Dr. Stewart and those of Drs. Evans, Pennington, and others, we find that we were mistaken, and we have decided to withdraw our certification of these milks in the future, at least our certification without examination.

REVIEWS.

On Professional Education, with Special Reference to Medicine. By T. Clifford Allbutt, M.A., M.D., F.R.S. London: Macmillan & Co., Lim., 1906.

Whatever else may be said of Dr. Allbutt, he cannot be accused of British insularity. He freely criticises the excessive conventionality of English public schools, their failure to encourage independent thought, and their tendency to encourage the English school-boy, during the plastic period of life, to shape his actions, pursuits, and mode of thought after what he calls "the monotonous pattern of Smith Major." He also touches on the question of athletics; not to disparage them for themselves, but to protest against the publicity of athletic sports, at least in secondary schools. While the subject of the address is chiefly the education of doctors, most of it is devoted to preparatory education in school and university. Facts are not the important things and are of no use until they are built into a theory. The world is moved by ideas, not by statistics; but for the purposes of mental discipline, scientific study is at least as effective as the so-called higher branches, history, the classics, pure philosophy, and ethics, and has the advantage that it can, and ought to be begun early.

Those who are unable to secure the advantages of a university training should devote the first two years of their five years' course chiefly to anatomy and physiology. These two subjects should be taught broadly, not for the purpose of imparting information only, but to train the student in accuracy of observation and logical deduction, and incidentally in the manual dexterity and resourcefulness which are essentials in the equipment of a practical doctor. Chemistry, while equally important, is too large and abstruse a subject to be treated in other than a practical manner as an aid to diagnosis, because the necessary time cannot be spared from the all-important subjects of anatomy and physiology and later of clinical medicine. In this connection and also in speaking of postgraduate work, Dr. Allbutt makes a strong appeal to the managing boards of hospitals for a greater liberality in opening their doors to students of medicine. "Every hospital, large or small, if its work is to thrive, and its patients are to reap the benefit, should be open to students. In teaching his pupil, the teacher educates himself—

reveals to himself his own latent capacities." In the later paragraphs of the address Dr. Allbutt gives a brief review of some of the great achievements of modern medicine and an optimistic forecast of its future, and devotes his closing remarks to a discussion of the practical side of the physician's life and certain general ethical questions. R. M. G.

PREVALENT DISEASES OF THE EYE. By SAMUEL THEOBALD, M.D., Clinical Professor of Ophthalmology and Otology in the Johns Hopkins University, Baltimore. Pp. 551. 219 illustrations and 10 colored plates. Philadelphia and London: W. B. Saunders Co., 1906.

THIS book has been prepared especially for the general practitioner. Its purpose is to acquaint him with all that can be known of ophthalmic practice without the ophthalmoscope, and without insistence upon the practical methods of measuring the optical conditions of the eye, or latent muscular anomalies; not that the author underestimates the importance of these fundamental conditions, for he is constantly and properly insisting upon them, and the imperative need of attending to them in the various diseased states in which they are a factor. What he wishes the reader to know, besides a general comprehension of what these errors are, and the signification of the terms describing them, is not necessarily how to correct them himself, but what conditions call for their correction.

With what success has the task been accomplished? There can be but one reply. The author has done so well that it is difficult to see how the book could be materially improved. The descriptions of such diseases as can be recognized by careful inspection, including oblique illumination, palpation, etc., are so clear that every medical reader should be enabled to profit by their perusal and apply the knowledge so gained to actual practice. Even of those diseases which require the use of the ophthalmoscope for their certain recognition (retina, choroid, optic nerve, etc.) the account is so lucid that the practitioner who is unacquainted with that instrument will be frequently enabled to form probable surmises, and will at least read with intelligence the report sent him by the specialist to whom he has referred the patient; for one of the main purposes of the author—in which he has admirably succeeded—is to teach the general practitioner which diseases of the eye he may treat himself and which require the services of a specialist.

But it would be a grave mistake to regard the book as of service to the general practitioner only. It will be found a useful standard of reference for the specialist as well. We surmise that it will find its highest appreciation in the hands of those practitioners who, while not specialists, are paying particular attention to the eye and who have perhaps attended postgraduate courses upon that subject.

In a large work like this it would, of course, be strange if perfect unanimity of opinion should exist upon all of the numerous subjects treated therein. Like all who have strong convictions of their own, the author sets forth certain views more or less individual, such as a supposed "trophic" influence in the causation of certain severe forms of iritis, the use of very large probes in the treatment of stricture of the nasal duct (of which procedure he has long been an advocate), the effect of accommodative strain in causing a special form of choroiditis, "miliary" choroiditis, the existence of subnormal accommodative power, etc. We read with some surprise of the author's extreme preference for argyrol and protargol as against nitrate of silver in purulent conjunctivitis. We are under the impression that the organic salts of silver, the favorite of Darier, had failed in this country to sustain the reputation he had given them. Is it true that interstitial keratitis "occurs only as a result of inherited syphilis?" If our memory serves us right, Hutchinson himself made no such exclusive claim as to the luetic origin in all cases. To our greater surprise and some regret we read that the author is inclined to be somewhat vacillating upon the necessity of a cycloplegic in measuring the refraction in young subjects; he says: "the employment of a cycloplegic introduces (in many instances) as many x qualities as are eliminated through the suppression of the power of accommodation," etc. We should like to have the practitioners told that a cycloplegic is necessary in every patient under forty years, unless some decided contraindication is present—of which there is but a single real one, glaucoma or suspicion of glaucoma.

We cannot leave this important work without a tribute to its excellent style and clear English. There is not an obscure sentence in the book. We have no doubt that it will be productive of a great amount of good. We hope that it will serve as an incentive and model for specialists in other branches of medicine to place before the general practitioner as clearly as this does for the eye, what he can and should know of the different specialties.

T. B. S.

SYPHILOLOGY AND VENEREAL DISEASE. By C. F. MARSHALL, M.D., M.Sc., F.R.C.S., Late Assistant Surgeon to the Hospital for Diseases of the Skin, Black Friars, London. New York: William Wood & Co., 1906.

THIS is a book of five hundred pages, octavo size, concisely written, original, and of interest alike to the physician and the surgeon. It is not a text-book in the usual acceptation of this term, but is written with the view of stating tersely all that is known on the subject, backed by the author's personal experience. The topics covered, although in some instances briefly disposed of, are numerous and varied, syphilis being considered as it involes the skin, the circulatory, alimentary, respiratory, genitourinary, osseous, and nervous systems, as well as the eye and other special organs. It will thus be noted that the subject is handled in a systematic manner. In further confirmation of this observation reference may be made to the interesting chapters on the systematology of heredo-syphilis, heredo-syphilis of the nervous system, and syphilis of the third generation. All the matter pertaining to these topics is presented in such a concise and lucid manner as to command attention and impress the reader favorably. At the close of each chapter is a list of references to the works referred to in the text. While the book, as already stated, is not large, judged by the size of most books of the day on this or allied subjects, it is evident, nevertheless, that much thought, time, and study have been devoted to the diseases considered, and that the principal authors of the time, as Fournier, Finger, Neisser, Balzer, and Jadassohn, have been wisely consulted. Gonorrhœa, in the male and in the female, receive separate handling, being viewed from both a practical and a scientific standpoint, and in a style quite different from that usually met with in literature. That the author, in common now with most physicians, thoroughly appreciates the gravity of gonorrhœa in its consequences in the female is borne out by the statement that it is the cause of a large proportion of the diseases of women. Syphilis and gonorrhœa are regarded as probably the oldest diseases which have afflicted mankind; and syphilis is believed *par excellence* the hereditary disease, and may be transmitted to the third generation. A number of reasons are tabulated for regarding Spirochæta pallida (Schaudinn) as the pathogenic agent of syphilis, the only fact wanting to prove the pathogenic nature of this organism being the absence of cultures, all attempts at culture (up to the present time) having failed in the the case of spirochetes, spirilla, and trypanosomes. Spirochæta pallida is difficult to find, and cannot be shown in all cases. Metchnikoff failed to find it in 26 per cent. of the cases he examined. The reviewer had the opportunity of studying with Schaudinn some of his sections, and can bear testimony that often these organisms in a given specimen are few and far between. The various methods of treatment of syphilis are briefly but satisfactorily considered, the author expressing himself in favor of the use of metallic mercury, either in the form of pill, gray-oil injections, or inunction. There does not appear to be, according to the author, any advantage in using other preparations, except in cases which are refractory to metallic mercury. Iodides should be used during the course of treatment according as indications arise for mixed treatment.

The book as a whole may be commended for its simplicity and at the same time for its scientific and practical aspects. Its teachings are sound and progressive, and the subject is presented in an interesting and readable style. We bespeak for it a cordial reception by the profession. L. A. D.

THE DYNAMICS OF LIVING MATTER. By JACQUES LOEB. New York: The Columbia University Press, Macmillan & Co., agents, 1906.

THE whole biological world, a large proportion of the members of the medical profession, as well as others interested in natural science and, indeed, many of the laity throughout the world have become very familiar with the main features of some of Professor Loeb's most striking contributions to biology; but the present volume makes available for those interested in such matters all the main details of his own work, his personal studies being made the basis of a most fascinating discussion of the subject under consideration, and being joined together by numerous and interesting references to the work of others upon similar lines. One would have difficulty in finding a book containing a greater amount of suggestive thought and experiment. Indeed, to one that has been only partially familiar with the author's work, it is amazing to see the many evidences of brilliant thought and of fertility in experiment. He takes up first, in the series of lectures that constitute the book, the general chemistry of life phenomena, giving an exceedingly lucid brief discussion of the most conspicuous recent work on enzymes in their various relations to life. He then considers the physical constitution of living matter; referring, for example, to foam-structures and emulsions, to the colloidal character of living matter and its bearing upon biological problems, to surface-films, to osmotic pressure, to the antagonistic effects of salts, etc. His next lecture refers to the physical manifestations of life, discussing the physico-chemistry of muscular contraction, cell division, and the origin of radiant energy and of electrical phenomena, etc. Next he takes up the role of the electrolytes, and the effects of heat and radiant energy upon living matter; considers heliotropism, and other facts concerning tropisms and related phenomena; and finally discusses fertilization, heredity, and regenerative processes, including, in these latter chapters of the book, a discussion of his remarkable work upon artificial parthenogenesis and the artificial production of hybrids, together with many other points of intense interest. Like many another brilliant investigator, he looks at some questions from the standpoint that particularly interests himself, and occasionally does not give quite enough credit to the other side of the question. Such enthusiasm, however, is often one of the most important factors in producing new knowledge; and it is unfortunate only so far as it occasionally leads the imperfectly instructed reader into too free an acceptance of some points that are still merely suggestive, rather than established. D. L. E.

A Text-book of the Diseases of the Ear, Nose, and Pharynx. By B. D. St. John Roosa, M.D., LL.D., and Beaman Douglass, M.D. New York: The Macmillan Co., 1905.

The most distinguishing features of this work are to be found in its thoroughness and completeness of scope, and in the common sense with which its authors deal with the problems of rhinology and otology. Both features are particularly noticeable in the portions of it which deal with the matter of treatment, and as methods of treatment are given more attention than any other aspects of their subjects, the authors are to be congratulated upon their achievement. Anatomy and pathology are but briefly dealt with compared with the amount of space allotted to the various measures calculated to give relief. We cannot but feel that this is a great advantage in a work of this character. So often such text-books are filled with lengthy descriptions of pathological appearances which are of but little value to the reader, and only serve to make the volume of greater bulk. Although a knowledge of the pathological conditions underlying diseases of the nose, throat, and ear is as absolutely essential to their proper treatment as it is to that of diseases elsewhere, nevertheless in a text-book commended for the use of students, or of practitioners, it is somewhat fruitless for the authors to exhibit their pathological knowledge to the exclusion of the practical deductions which they draw therefrom, and which are what the reader chiefly seeks in every book.

Every aurist of experience knows, or should know, that there is much that can be done to alleviate the sufferings of those who have chronic catarrhal ear troubles, and he also is unfortunately aware of the fact that much can be done to accelerate their progress by measures injudiciously directed toward their relief. We feel that Drs. Roosa and Douglass have struck a happy medium between advising too assiduous treatment and, on the other hand, encouraging as fully dangerous a neglect.

It is to be regretted that the illustrations are not of better quality; most of them are reproductions of old plates, and not at all the kind one expects in such an up-to-date book. As, however, so many of the modern books depend almost entirely upon the attractiveness of the illustrations, regardless of the value of the text, this feature may be passed over with slight regret. F. R. P.

Operative Otology—Surgical Pathology and Treatment of Diseases of the Ear. By John Clarence Blake, M.D., Professor of Otology in Harvard University, and Henry Ottridge Reik, M.D., Associate in Ophthalmology and Otology, Johns Hopkins University. New York and London: D. Appleton & Co., 1906.

In the preface to this book the authors make the rather comprehensive claim that it is written for "the use of the student, the general and the special practitioner." After a careful reading we cannot but grant that they come very near, if not quite up to, the fulfilment of their promise. For the student the special value of this volume lies in the plainness with which it describes points in technique which are usually only learned by most beginners in otology after some hard experiences. The practitioner will find its pages exceedingly helpful in time of trouble, because of the fulness with which the authors deal with the many aural complaints which generally fall to the lot of the general practitioner, at any rate in their incipiency. To the specialist the value of the book will be found to consist in its up-to-dateness; all the freshest and newest subjects of interest in otology are here handled with a proper zeal qualified by a due discretion. An excellent section of the work is that devoted to "Adventitious Aural Surgery," in which the authors describe adenectomy, lumbar puncture, subcutaneous and intravenous injections. The descriptions of the various intratympanic and mastoid operations are excellent. There is no section of the work devoted especially to diseases of the Eustachian tube, and in fact that important *via dolorosa* receives but scant mention throughout the volume. The illustrations are especially commendable. Most of them have been made expressly for the book, and it is a pleasure to welcome a text-book on otology which is not filled with drawings purloined from Politzer and Gruber. F. R. P.

Photoscopy (Skiascopy or Retinoscopy). By Mark D. Stevenson, M.D., Ophthalmic Surgeon to the Akron City (Ohio) Hospital. Illustrated. Pp., 126. Philadelphia and London: W. B. Saunders Co., 1906.

This is a somewhat unequal book; many of the explanations of the theoretical portion are obscure; happily the practical directions for employing the test are in the main clear and precise. They should enable the practitioner who is conversant with ophthalmological methods to begin and carry to a successful issue the study of the practical application of the shadow test without an instructor. This we regard as no slight merit, and is the main reason why we welcome the appearance of the work, since it will, no doubt, succeed to a greater or less extent in calling the attention of physicians, engaged in refraction, to the great value of skiascopy as the best practical objective test for the determination of the refraction of the eye.

There are naturally certain legitimate differences of opinion as to some of the injunctions prescribed, as when, in the estimation of astigmatism, preference is given to the measurement of each meridian by a sphere instead of by a combination of sphere and cylinder. This latter is, indeed, mentioned, but little is said of its special advantages for finding the true axis by the change in the direction of the band when the cylinder is wrongly placed. We also think that the reasons for working with the light close to the (plane) mirror admit of much stronger emphasis than is placed thereon by the author—surely the argument therefor is better than the preference each is inclined to give to his "own ophthalmoscope rather than any other." To paralyze the accommodation, homatropine hydrobromate is recommended in the strength of 4 grains to the ounce; thrice this strength is necessary to insure complete paralysis of the accommodation. The choice of the term anterior focal point for the point of reversal is unfortunate; the former term is applied to the most anterior of the cardinal points of the eye.

These lapses, however, as we believe them to be, are comparatively unimportant, where there is so much that is commendable. We have pointed them out with the hope that the author may subject all portions of his book to a careful revision when the time comes for another edition. We are so impressed with the value of the shadow test that we are anxious that everything connected therewith shall be presented clearly and free from blemish.

T. B. S.

Chirurgie Oto-rhino-laryngologique. By Georges Laurens, M.D., of Paris. 470 illustrations. Paris: G. Steinheil, 1906.

This large work in two volumes is one of a series of treatises on operative surgery published under the editorship of Profs. Berger and Hartmann. It may be regarded as a thoroughly modern exposition of the latest French views on the surgery of the ear, nose, and throat. We have rarely met with a clearer description of the complicated operations upon the ear than this work presents. This is not only true of the various operations in the mastoid region, but also of the intratympanic procedures. The sections devoted to intranasal and laryngeal operations are also excellent. The work is copiously illustrated with handsome plates, which are not only good artistically but really helpful in the elucidation of the text.

F. R. P.

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UNDER THE CHARGE OF

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The Clinical History and Significance of Clubbed Fingers.—EBSTEIN (*Deut. Arch. f. klin. Med.*, 1907, lxvii, 89) gives a good review of the literature of clubbed fingers. He states that this our usual English term is a poor one and prefers the more accurately descriptive term used in Germany and France, "drumstick fingers." He thinks the term hippocratic fingers should not be applied to the condition, as Hippocrates described only the incurving of the nails and did not mention the characteristic thickening of the terminal phalangeal joints. Trousseau found thickening of the terminal phalanges, in a more or less marked degree, in nine-tenths of all subjects who had had definite signs of pulmonary tuberculosis for three months or over. In a large number of cases other than pulmonary tuberculosis, he observed the condition only twice, once in a girl with cardiac disease and once in an apparently healthy young man. Clubbing of the fingers is such a frequent occurrence in pulmonary tuberculosis that Trousseau says its presence has led to a diagnosis of the disease when other physical signs were equivocal. Later observers point out how frequently clubbing occurs in other conditions and Blandin summarizes as follows: We know that curvature of the nails occurs not only in phthisis, but in all chronic conditions associated with wasting. Heller, too, emphasizes the little diagnostic and prognostic significance attaching to clubbing of the fingers. In bronchiectasis the condition is particularly well-marked and the fingers are, as a rule, shorter and broader than in phthisis. The nails frequently show an ulnar deviation. The clubbing of the fingers comes in some cases with great rapidity, and Bamberger states that it has been noticed to develop from the time the previously odorless sputum becomes fetid. Gerhardt considers clubbing the earliest stage in a series of rheumatoid affections of the joints, common in bronchiectasis, and of the bone changes described by Marie. Clubbing also frequently follows empyema. Dening reports a unique instance of the development of typical clubbed fingers in a case of gastric dilatation followed by pyloric stricture from a healed ulcer. After operation the clubbed fingers returned to their normal condition. The French authors particularly call attention to the association of clubbed fingers and hepatic cirrhosis. The condition is most marked in the cirrhoses of children. Gilbert and Lerchoullet have collected 40 instances. In the cases of autotoxic enterogenous cyanosis described by Stokvis and v. d. Berg, clubbed fingers is one of the characteristic symptoms. The relation of the changes in the fingers to the pulmonary osteoarthropathy of Marie, or, as Sternberg more correctly terms it, toxic osteoperiostitis ossificans, has given rise to much discussion; 75 per cent. of the cases of osteoperiostitis occur in association with pulmonary lesions—tuberculosis, bronchiectasis, and other purulent affections of the lungs and pleura, and it is generally conceded that clubbing of the fingers represents the first stage of the condition. In myxœdema and pulmonary tumors clubbed fingers occasionally occur, and Swoboda has recently called attention to their frequent presence in rachitic children with deformities of the chest causing restriction of respiratory movements. Next to disease of the lungs clubbing of the fingers is most common in cardiac lesions; unusual in acquired, but very frequent in congenital disease. It occurs particularly often in pulmonary stenosis and in defects of the septum. Of great interest are the cases of clubbing restricted to one hand. The condition is unusual, and in the few reported instances has always been associated with thoracic aneurysm. Shrinkage of the sac has been followed, in some cases, by disappearance of the clubbing. The pathology and etiology of the condition is not well understood; Ebstein gives at some length the various views prevailing.

Erythema Exudativum Multiforme and Nodosum of the Mucous Membranes and Their Relation to Syphilis.—Trautmann (*Münch. med. Woch.*, 1906, liii, 2101) calls attention to the importance of making an accurate differential diagnosis between syphilitic and erythema multiforme and drug eruptions of the mucous membranes of the mouth and upper air passages. In 39 cases of erythema multiforme he found a lesion of the mucous membrane in 20 cases, a mucous membrane eruption subsequent to the skin eruption in 4 cases, a primary mucous membrane eruption with subsequent skin lesion in 11 cases; a solitary mucous membrane eruption in 4 cases. Many of these occurred in patients who had previously had syphilis, and had been subjected to repeated and heroic mercurial treatment without benefit and, indeed, often with a good deal of harm. The erythematous lesions in the mouth undergo maceration and destruction and come to resemble very closely specific lesions; the deception is particularly imposing when there is an associated papular erythema of the skin or the nodes of erythema nodosum along the tibia. In the latter instance the diagnosis is especially difficult, as Hoffmann and Mauriac have described a syphilitic form of erythema nodosum which is clinically hardly distinguishable from it.

Trautmann believes that any non-specific erythema multiforme may occur synchronously with true syphilitic lesions of the skin, and the two must not be referred to a common cause. The curious herpetic eruption described by Fournier, the herpes recidivant Fournier, is preceded in 96 per cent. of the cases by syphilis. Trautmann considers the syphilis, however, merely a predisposing agent, just as tuberculosis so very frequently occurs in diabetes. Mucous membrane eruptions due to mercury are not uncommon. They occur usually in an area between the base of the tongue and the larynx. They are, of course, aggravated by pushing antisyphilitic treatment. The 7 cases Trautmann reports are interesting and important. In all there were lesions of the mouth or larynx in patients giving a history of previous lues; all had stubbornly resisted antisyphilitic treatment; all responded favorably to salicylates.

The Roentgen-ray Diagnosis of Unusual Heart Lesions.—DENEKE (*Deut. Arch. f. klin. Med.*, 1907, lxvii, 39) reports two cases in which a study of the heart shadow was of importance in diagnosis. The position, form, and size of the heart have been very carefully studied, but Deneke thinks too little attention has been paid to the intrinsic movements. In his first case, an instance of Stokes-Adams' syndrome, the partial heart block could be beautifully demonstrated by watching the movements of the auricles and ventricles. In a case of congenital heart disease a probable diagnosis of transposition of the large vessels and a defect in the interventricular septum was made, and the assumption was verified by autopsy. Fluoroscopic examination aided greatly in arriving at this conclusion. Normally the right border of the cardiac shadow is formed by the right auricle, and this portion of the heart makes only faint fluttering contractions, while the left border formed by the left ventricle is seen to contract forcibly. In the reported instance both borders contracted synchronously and forcibly and the whole heart was drawn together with the strong, sustained impulse. Evidently, there was a greatly hypertrophied right ventricle pushing the auricle upward or backward. As pulmonary stenosis and patent ductus Botalli could be excluded by other physical signs, a defect in the interventricular septum was diagnosticated.

Fever.—BEITZKE (*Berl. klin. Woch.*, 1907, xxxiii, 110) reviews the recent contributions to the pathology of fever and summarizes the present state of our knowledge. He points out that a definition of fever is itself very difficult and no two authors exactly agree. It is hard to distinguish which are the manifestations of the fever, which of the infection. All varieties of fever have in common an increased body temperature and an increased metabolism, the other manifestations being partly due to this, and partly independent of the fever. The increase of body temperature depends upon an increased heat production and a decreased dissipation. In a normal person these two factors are so nicely adjusted that even under extreme circumstances, such as excessive heat production with muscular exertion and great variations in the external temperature, the body temperature remains unchanged. This balance is broken in fever, but the two factors are varyingly disturbed. During the rise of temperature heat production is particularly increased, often more than at the height of the fever, especially if there is a chill. Heat dissipation is usually decreased, but often remains unchanged, and at times is even increased. During the height of the fever production and dissipation are both increased; the latter, in many cases, over 50 per cent. But they bear no direct ratio to one another, and, as a rule, run as separate curves more or less widely separated. The percentage of heat dissipation by retention, conduction, and evaporation retains usually a normal relation, while during muscular exertion 78 per cent. of the loss occurs by evaporation. In fever with great emaciation, production and dissipation both sink, production being often very low, but fever still persists because dissipation remains below it. As the temperature falls production always falls, but dissipation shows a varying dissipation. During a crisis it comes down rapidly, during lysis very slowly. It may even be subnormal, and the two curves of production and dissipation slowly approach one another.

The origin of the increased heat production is the heightened metabolism. It is true that the temperature itself may be responsible for a part of this increase, but the metabolism of fever is far greater than that caused by simple overheating and, as a rule, it begins before the onset of the fever. The relation of the carbonic acid secretion to the oxygen inhaled and of the carbon excretion to the nitrogen intake remain normal unless there is great emaciation. The increased metabolism affects principally the albuminous substances, the nitrogen excretion increasing from 50 to 150 per cent. The albumin is usually burnt to its end product, urea, but earlier stages of the oxidation often appear in the urine. Qualitatively the reduction reactions are probably the same as in health. It is difficult to ascertain the degree to which carbohydrates participate in the increased metabolism. It has been shown that carbohydrates may be derived from albumin, and some authors see in them the source of energy of albumins. At least all existing carbohydrates are rapidly burned. Fat is consumed to no greater extent than in any condition of inanition. In all fevers there is a marked retention of water and of inorganic salts, particularly of chlorides.

The place where the increased metabolism principally occurs is disputed. Krehl, Rolly, and others designate the liver; Senator, Richter, and Aronsohn the muscles. The reasons in favor of the latter view are, in health three-fourths, in fever two-thirds, of the carbonic acid is formed in the muscles. In fever the temperature in the muscles rises from 3.76 to 4.48°; in the liver only from 2.3° to 2.88°. In fever the urine is rich in creatin and other products of muscle metabolism. The increased activity of the heart and respiratory muscles alone accounts for one-twentieth of the whole heat production.

Most fevers are caused by the circulation of free toxins either of bacterial or inorganic origin or by the absorption of organic poisons from abscesses or blood clots. Krehl believes these may act by directly stimulating metabolism. In other instances, however, fever follows cerebral compression, and in these cases a direct influence upon the heat-regulating centre must be assumed. Aronsohn and Sachs were the first to produce fever by irritation of the corpus striatum. They do not believe in a direct albumin disintegration by the toxins and they think that all fevers are due to changes in the nervous system affecting the heat regulating centre. That fever is the expression of a beneficial reaction of the body is gaining ground. Of itself it could be harmful only if it arose to a very high degree, when the same dangers are to be feared as in insulation. Such an occurrence is rare. The same is true of the increased metabolism which becomes a danger only when there is great inanition. Every physician knows what a bad prognostic sign little or no fever is in severe septic or typhoid infections. Similarly a fall of temperature after artificial animal inoculations is a bad omen. By high temperatures bacteria and their products are partly destroyed, partly rendered harmless. The reaction between toxin and antitoxin takes place better at an elevated temperature.

Muscular Cirrhosis of the Lungs.—Davidsohn (*Berl. klin. Woch.*, 1907, xliv, 33) bases his paper on an autopsy on a man who died of septic endocarditis. The man is said to have suffered for some time with some variety of pulmonary inflammation. The lower lobe of the left lung was so fleshy in appearance that the term carnification was the only one that would fit the microscopic description. That this carnification was due to the presence of smooth muscle fibers was first suggested by the brown appearance of the cells in Van Gieson's stain. The fibers were gathered in bundles running in all directions and without special arrangement, resembling very much a uterine myoma. The pleura covering the lobe was changed into a thick fibroid mass without any muscle cells. The first mention of this pathological condition is by Buhl, in 1873, his description corresponding closely to that of Davidsohn, and its rarity is apparent from the statement of Orth that he himself has never seen an instance. There is now abundant evidence to prove the existence of smooth muscle fibers in normal lung tissue, and Orth notes that they may be much increased in number in brown atrophy. There is nothing in favor of assuming a metaplastic transformation of the connective-tissue cells into smooth muscle fibers. The possibility of the condition being a metastatic myoma or an angiomyoma arising from the vessel walls must be considered. Against the first may be urged that the process is diffuse and not at all like a tumor, and the entire absence of large numbers of lumina of bloodvessels excludes the second. The striking resemblance to the latter condition noted at first glance is shown to depend upon the presence of numerous collapsed alveoli. It seems most probable that the muscle fibers arise by the division and proliferation of preëxisting cells. Davidsohn suggests that the term carnification be reserved for these unusual instances of smooth muscle overgrowth.

Eosinophilic Intestinal Disease.—Neubauer and Staubli (*Münch. med. Woch.*, 1906, liii, 2380). The occurrence of eosinophiles in certain inflammations of the mucous membranes is accompanied by the formation of Charcot-Leyden crystals and frequently by an increase of eosinophiles in the blood. Evidence points to the Charcot-Leyden crystals arising from the eosinophile cells, and their occurrence has the same significance as finding eosinophile cells. The increase of eosinophiles in the blood is not a constant feature and in some instances they may be diminished in number or even absent. The occurrence of Charcot-Leyden crystals in the stools was first observed in ankylostomiasis and later in other intestinal parasitic infections. Eosinophiles rapidly disintegrate in the bowel, and although they may be missed in the stools there is often an increased number in the blood. This occurrence of eosinophilia with intestinal parasites is so striking that Lichtenstirn has considered them pathognomonic. But later authors have found them to occur in other conditions. Neubauer and Stäubli report cases of acute diarrhœa with marked constitutional symptoms, finding in the stools of the first Charcot-Leyden crystals, and in the second, eosinophiles. During the height of the attack the eosinophile cells had completely disappeared from the blood, returning with abatement of the symptoms. Analogously, the absence of eosinophiles in the blood has been noted in the acute stage of bronchial asthma, and it has been suggested that the supply is drained by the sudden local demand. Dock has shown that eosinophiles may be present in the stools of patients with amœbic dysentery. In the third case reported by the author, one of acute dysentery, they were likewise found, although amœbæ were absent and no typical dysentery bacilli could be cultivated. In the last three cases reported an interesting affection of the lower bowel is considered, which the authors believe is distinctive enough to be classed as a separate clinical entity. All are remarkably similar, occurring in young adults with symptoms of severe diarrhœa and discharges of blood and mucus. Charcot-Leyden crystals were found in the stools of all and numerous well-preserved eosinophile cells in two. Intestinal parasites as well as specific bacteria (dysentery bacilli, tubercle bacilli, gonococci) were absent. The rectal examination through the proctoscope showed an intensely red, loose, mucous membrane with numerous small, yellowish-white areas of exudate which could be easily scraped off and under the microscope were seen to be made up of eosinophile cells and granules and Charcot-Leyden crystals. After scraping off the exudate a deeply injected bleeding erosion remained. There were no ulcers and no cicatrices. Two of the cases showed a distinct increase of the eosinophiles in the blood. Helber, in 1905, described a disease similar to this as sigmoiditis chronica granulosa, but as he says nothing about eosinophiles, the two conditions cannot be definitely identified as the same.

SURGERY.

UNDER THE CHARGE OF

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A Contribution to the Study and the Treatment of Impassable Noncancerous Strictures of the Œsophagus.—Gross and Sencert (*Revue de chir.*, 1907, xxvii, 1) say that when one has failed, after repeated and careful attempts, to pass through a stricture from above downward, an examination by the œsophagoscope is necessary. Often the stricture will thus be rendered passable, by exposing its upper orifice to the view of the surgeon. Only exceptionally will this method fail. Eccentricity of this orifice is the chief obstacle to blind catheterization. Once the dilatation has been begun it can be carried out without the aid of the œsophagoscope. When a very old and resisting stricture cannot be overcome by progressive dilatation, a metal instrument of the same caliber as the fine dilating bougie should be passed. A thread is tied to its upper end and brought out of the mouth. The instrument is left in place with little or no disturbance to the patient, and is withdrawn by simple traction after twenty-four hours. Ordinary dilatation may then be carried on, or a metal instrument of larger caliber may be introduced by the aid of the œsophagoscope, or this may be continued with instruments of larger caliber until the normal caliber of the œsophagus is obtained.

If the examination by the œsophagoscope reveals a diverticulum or prestrictural pouch, with more or less inflamed and fragile mucosa, threatening postoperative dangers, the method of treating the stricture should be changed. Two varieties of cases present themselves: those in which a gastrotomy has already been performed, and those in which it has not. If a fistula exists, retrograde catheterization may be tried, and if one has the good fortune to pass the cardiac end of the œsophagus, he can practise dilatation immediately, without recourse to the method of von Hacker. Unfortunately this will often be impossible.

When there is no gastric fistula, Delagénière has employed a large gastrotomy in order to pass the cardia and the stricture with a sound. Dilatation by von Hacker's method is then carried out. Gross and Sencert say that this operation exposes the patient to considerable danger from peritonitis and to the possibility of failure to pass the stricture with the dilating instrument. A permanent gastric fistula is then the only salvation of the patient. In such cases the authors propose an operation which they have studied on the cadaver. A median laparotomy above the umbilicus is performed; the stomach is exposed and seized at a point on the anterior surface, about 2 cm. below the cardia. A purse-string suture passed through the external coats of the stomach without going through the mucosa is made to circumscribe here an area 1.5 cm. by 2 cm. in diameter, the ends of the suture being left untied. An opening is made in this area into the stomach and an œsophagoscope introduced, when the purse-string suture is tightened about the tube, preventing the soiling of the peritoneum with stomach contents. The tube being cleansed, the cardia is sought and with care the lower end of the stricture is found. Simple retrograde dilatation is then carried out with the aid of the œsophagoscope. The Gross and Sencert feel certain that this method of retrograde catheterization is a better means of finding the lower end of the stricture than the large gastrotomy of Delagénière. If all these methods fail, then the major operations—external œsophagotomy, œsophagectomy, œsophagogastrotomy—may be considered, but only after repeated failure by all the preceding methods.

The Appendicitis Question.—Haim (*Zentralb. f. Chir.*, 1907, xxxiv, 33) made a bacteriological examination in cases of streptococcic appendicitis, the results of which he discusses briefly and with caution as to their general reliability. He hopes that they will encourage the collection of statistics of the appendicitis cases operated on in the acute stage, from which we may settle many questions arising concerning them, and obtain a clearer conception of their nature. From his observations he concludes that the severe, diffuse, streptococcic cases occur most frequently in the spring, in the months of March and April, and in the fall months of October and November, and that they are very rare at other times. Again, certain symptoms are more common in certain groups, as cases with herpes, or with icterus, etc. The colon bacillus, which produces a milder form, seems to be equally active throughout the year. The etiology seems to vary in different countries, in the northern countries the bacillus coli and the pneumococcus prevailing; and in Germany, France, and probably in America, the bacillus coli and streptococcus; while in Italy and England usually the bacillus coli. This may explain the great difference in mortality of different authors. The streptococcic cases occur by preference in children and adolescents, the milder forms in older people. The prognosis in streptococcic cases is better in the young than in the old, in whom they are almost always fatal. The prognosis is, of course, dependent also on the time of operation: the earlier the operation the better the prognosis. The intermediate stage here offers no advantage, so that no delay in operating should be advised. The diagnosis can be made only by the symptoms. Greater material placed at our disposal will probably support these observations and develop new points. In all cases the bacteriological examination should involve the contents of the appendix and the resulting exudate.

Three Cases of Tumor of the Bladder without Hematuria.—Guizy (*Annales des maladies des organes genito-urinaires*, 1907, xxv, 103) reports one case in a married woman, aged fifty years, without children, who suffered from very frequent urination, cloudy urine, and at times complete retention. The cystoscope revealed an enormous papillomatous tumor with three long prolongations, occupying a large part of the superior zone of the anterior wall. There was no hematuria whatever. By a suprapubic cystotomy he removed the tumor, which occupied the greater part of the cavity of the bladder. Two years later, the patient remained completely cured.

In the second case—a married man, aged fifty-eight years, suffering from frequent and painful micturition—the cystoscope revealed a tumor with a large pedicle, occupying the mediosuperior zone of the anterior bladder wall. The urine was cloudy and ammoniacal, with a few very fine sanguinolent filaments. The patient never had hematuria, not even a drop of blood. Nothing abnormal could be determined in the renal regions, but the liver was enlarged, irregular, indurated, and painful. Death occurred after some weeks, with a carcinomatous cachexia.

The third case was in a woman, aged forty-one years, without children, who suffered from frequent and painful micturition. The exploring sound developed an extreme sensitiveness of the vesical mucosa. The cystoscope showed a papillomatous tumor, which was removed by a suprapubic cystotomy and found to have a very large and short pedicle, attached to the superior part of the anterior wall of the bladder. There was no hematuria. The question arises as to whether the attachment of all three of these tumors to the anterior wall of the bladder explains the absence of hematuria.

Tennis Elbow.—Preiser (*Zentralbl. f. Chir.*, 1907, xxxiv, 65) says that there is a characteristic injury resulting from tennis playing, just as we have a fracture of the radius in chauffeurs and a fracture of the malleolus in foot-ball players. The tennis injury has already been referred to by Clado, who considered that it is due to a rupture of some of the fibers of the supinator brevis muscle. Bähr, in reporting two cases, says that the injury was traceable to a forcible movement of the forearm in pronation, and that the head of the radius was very painful to pressure He thought that the degree of pain suggested a periostitis, but that there might be rupture of a ligament.

Preiser has observed two cases which convinced him that the condition was not the result of a tearing of the supinator brevis fibers. His patients were strongly built men, who during the tennis season developed gradually a pain in the right elbow. Finally tennis playing became a punishment and complete extension impossible. Effusion in the elbow could be recognized. By pressure of the finger during pronation and supination, as well as in tennis playing, the pain was especially localized to the palmar surface of the forearm. Flexion became painful and limited toward its completion, and extension was possible only to an angle of 160 or 165 degrees before it became painful. Passive extension was complete with pain, but movement between the painful limits was free and free of pain. The *x*-rays showed no fracture, while the effusion in the joint indicated, according to the author a process in the joint capsule.

Preiser explains the localization of the injury by the origins of the supinator brevis and brachialis internus muscles. Each has a part of its origin from this portion of the annular ligament, the latter muscle being a strong flexor and pulling upward on this part of the capsule. The supinator brevis pulls in the opposite direction, that is, downward. The result of long-continued violent contraction of these muscles would be to produce a gradually increasing chronic inflammation at this part of the capsule. Those parts of the play necessitating strong flexion and supination are evidently responsible for the lesion. The condition yields stubbornly to treatment. The first indication is to forbid tennis playing. Massage, especially friction of the infiltrated capsule, brings greatest relief. Bathing of the elbow twice daily in hot water should be ordered.

Plastic Repair of the Peritoneum with Isolated Omentum.—Girgolaff *Zentralbl. f. Chir.*, 1907, xxxiv, 113) in this contribution answers the criticisms of Springer, who compared Girgolaff's experimental results with his own and found Girgolaff's conclusions too sweeping. According to Springer's experiments, extensive peritoneal adhesions occur, and absolute protection against perforation of the intestine does not occur, if the insecure sutures are covered with an isolated portion of the omentum. Against the first statement of Springer, Girgolaff can only refer to the results of his own work on the stomach, large intestine, and bladder, where no adhesions resulted. He says that an absolutely sure preventive against adhesions does not exist. In his work on the small intestines he found that when special efforts were made to obtain thorough asepsis and hemostasis the adhesions were very few and unimportant, but if an inflammatory infiltration or a considerable hemorrhage occurred, then the resulting adhesions were considerable. Girgolaff did not see any long bands which might have led to strangulation. It is noteworthy that when wounds of the upper and anterior surface of the liver were covered with pieces of omentum no adhesions developed, but they did occur on the under surface where the blood accumulated. Tuffier has employed isolated omentum to cover a wound of the liver, and Lebreton to cover a wound of the spleen, both with advantage. To cover the proximal stump of the omentum, as in hernia operations, is always of value. Girgolaff has never seen adhesions develop from this. In regard to the effectiveness of the closure of a perforation by an isolated piece of omentum, Girgolaff refers to his own and Sundholm's results, and says that after making a permanent opening in the intestine, by suturing the mucosa to the peritoneum, he closed this opening by covering it with isolated omentum, and in no case did peritonitis result. If the omentum is rich in fat, the excess can be removed by scissors, thus increasing its ability to form adhesions.

Surgery of the Hand.—Van Hassel (*Annales de la Société Belge*, 1907, vii, 19) says that the recent law on the accidents of workingmen has made the surgery of the hand of much importance, because of the importance of this member to the laboring man. At the last meeting of the Surgical Society, Gyselynck and Lemoine presented a remarkable paper on this subject. In burns the writer has obtained his best results by irrigations and dressings with artificial serum. Burns of the third degree, however, require careful watching to prevent troublesome deformities from contraction, and when these do occur they require autoplastic operations. Punctured wounds are more troublesome than incised, because of the danger of deep infection and lymphangitis. When there are foreign bodies in the wound, Van Hassel employs cupping and the Bier bandage. In contused wounds, after thorough irrigation with bichloride solution, he proceeds immediately to bring together, as carefully as possible, all the divided tissues, torn periosteum, tendons, cellular tissues, in different planes, draining as well as the conditions will permit. In this way he tries to overcome the adhesion of the skin to the deeper layers by scar tissue. He immerses the part daily in the physiological serum, and irrigates freely with boric-acid solution. With this method he has been able to obtain a perfectly useful hand in cases in which, at the time of the accident, it seemed that amputation was necessary.

THERAPEUTICS.

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The Pharmacology of Digalen.—J. B. Reneau (*Revue thérapeutique*, 1906, lxxiii, 721) concludes a study of the pharmacology of digalen (soluble digitoxin) with the following statement: If digalen, from a chemical point of view, does not appear to be a definite substance and one differing from the other active principles derived from digitalis, its manner of action upon the organism, its toxicity, and its local effect would lead one to consider it a substance nearly kin to digitalin, possessing similar cardiotonic and vasoconstrictor properties which are susceptible of interesting therapeutic utilization by reason of its feeble toxic action, of its lack of cumulative effect, and its weakly irritant local action.

Ovarian Extract in the Obstinate Vomiting of Pregnancy.—G. Stella (*La semaine médicale*, 1906, xxvi, 523) believes that ovarian opotherapy possesses a true field of usefulness in the hyperemesis of pregnancy. The patients who he has treated by this means have been immediately relieved of this troublesome symptom, and his results have been so striking that he states that this form of treatment should always be tried before the advisability of emptying the uterus is considered. The dose prescribed corresponds to 45 grains of the fresh gland; in the most severe instances of vomiting this quantity may be progressively increased to twice this amount.

The Pharmacology of Veratrum.—Wood, Jr. (*Jour. Amer. Med. Assoc.*, 1906, xlvii, 2061), draws the following conclusions from experimentation with this drug: (1) Neither veratrin nor rubijervin is present in veratrum viride root in sufficient amount to be held responsible in any degree for the activity of the drug. (2) Protoveratrin, although an extremely poisonous substance, differs essentially in its effects from the veratrum rhizome, and, therefore, is not to be considered its active principle. Moreover, the proportions of it are too small to permit of its being considered the toxic ingredient. (3) Of the known proximate principles, jervin most nearly corresponds in its physiological action to the crude drug, but is present in too small amount in proportion to its toxicity to be considered the chief active ingredient. (4) The evidence, therefore, seems to be conclusive that there is in veratum viride some active substance as yet undiscovered.

The Preventive Treatment of Serum Disease.—W. d'Este Emery (*The Practitioner*, 1906, lxxvii, 754) discusses the prophylaxis of this condition, which is due to the injection of antitoxic sera and is characterized by fever, a rash of variant type, lymph-gland enlargements, and other symptoms. He says that Netter's preventive treatment, which consists in the administration of calcium chloride, is a decided therapeutic advance. The drug is administered in doses of 15 grains at the time of each injection and for two days subsequently. Among 114 cases treated thus, Netter observed 11 mild instances of serum disease, whereas in a control series of equal number the condition developed twenty times. These results are readily credible in view of the well-known fact, first shown by Wright, that many cases of urticaria are associated with a deficient power of coagulation of the blood and a diminished calcium content, and are curable by calcium chloride or lactate. Why the injection of serum should cause such alterations in the blood and what is the reason for the delay in the appearance of the symptoms are facts less clearly understood; possibly the formation of the precipitins, or of the precipitates they cause, may remove some of the calcium from the blood. If this is the case the serum disease might develop in a few patients only, that is, in those with a deficient calcium content in the blood.

Irrigations with Pulp of Pigs' Liver in Hepatic Cirrhosis and Tuberculous Peritonitis.—Desplats (*Jour. de sci. med. de Lille*, 1906, No. 30, p. 73) reports an instance of tuberculous peritonitis and cirrhosis of the liver, which when first seen presented general anasarca, dyspnœa, albuminuria, and hematuria. After being supervised for four months, during which time tapping was often necessary, irrigations of 10 ounces of a pulp made from pigs' liver were prescribed. In less than two months there was marked improvement, the hematuria was less, tapping was no longer necessary, and the masses in the peritoneum had decreased in size. The patient continued to improve rapidly and returned to work. Desplats reports a second similar instance in which just as remarkable a cure was attained.

The Treatment of Tuberculous Meningitis.—G. Reabold (*Münch. med. Woch.*, 1906, No. 35, p. 1709) advocates treatment by repeated lumbar punctures and the withdrawal of the cerebrospinal fluid. He reports a cure in a patient in whom bacteriological tests confirmed the diagnosis and in whom the operation was performed twenty-four times with the withdrawal of a total quantity of 574 c.c. of fluid. In his opinion the results of the treatment are most striking. It is followed by an almost immediate amelioration of the patient's condition, which is rendered progressive by means of daily withdrawal of fluid and the consequent relief of intraventricular pressure. It should not be forgotten that the fluid is likely to re-accumulate rapidly. In determining the indications for puncture the subjective condition of the patient and the evidences of intracerebral pressure, as manifested by headache, etc., should be taken into consideration. When the febrile movement disappears and the albumin content of the cerebrospinal fluid becomes diminished, the intervals between the punctures may be lessened.

Tuberculin Treatment.—Sahli (*Correspondb. f. Schweizer Aerzte*, 1906, Nos. 12 and 13, pp. 373, 417) states that, in his opinion, this form of treatment is, when the proper dosage is not exceeded, entirely harmless. All reaction should be avoided. In favorable instances the general condition becomes improved and the cough and physical signs are diminished. If fever is present the tuberculin treatment is contraindicated, since here, in Sahli's opinion, the system is already saturated with tuberculin. He also considers that mixed infections are unusual and that failures in treatment should rarely be attributed to this cause. He employs Beraneck's tuberculin, the dosage of which should be varied in accordance with the susceptibility of the patient. The usual number of injections is two per week.

Sodium Citrate in the Dyspepsia of Adults.—L. Lacheny (*La semaine médicale*, 1906, xxvi, p. 536) has, after numerous experiments, shown that this substance is potent in certain gastric disorders. He prescribes it in 10 per cent. aqueous solution, of which the dose is a dessertspoonful. This quantity is taken every five minutes until the gastric pain or nausea, which some patients so frequently exhibit after eating, has disappeared. He has achieved excellent results in a considerable number of patients by this means of treatment. In the most favorable instances, at least 15 grains of the citrate are necessary to relieve the symptoms, and resistant patients may be forced to take seven or eight times this amount to obtain relief. Cure, at least temporary, of the symptoms is usual, and if there is a recurrence the treatment may be repeated.

The Medical Treatment of Malignant Disease of the Stomach.—Einhorn (*The Postgraduate*, 1906, xxi, 1192) suggests that it is always wise to try antisyphilitic treatment in instances of gastric tumor, even though all symptoms point to the probable malignant nature of the growth, for although failure may occur in 95 per cent. of the patients, much will be accomplished if the other 5 per cent. are saved. In the management of true cancerous disease Einhorn considers it a mistake to inform the patient of the nature of his complaint; sedatives to relieve the pain are advised. Diet is an important question, and if the growth does not cause so marked a stenosis as to prevent the passage of fluids, a strictly liquid diet is to be prescribed, consisting of eggs beaten in milk or bouillon, oyster broth, calf's foot jelly, etc. Two or three quarts of milk daily will cause an increase in the patient's weight. If there is no stenosis a semisolid diet is advisable, consisting of farina with milk, pea-soup, etc. With pyloric obstruction operation should be advised. With regard to medicines fluidextract of condurango bark should be mentioned; it improves the appetite and is an excellent stomachic. Hydrated chloral acts as a sedative and prevents fermentation; it may be given in 3 per cent. solution, a tablespoonful three or four times daily Methylthionine hydrochloride seems to prolong life in some instances and may be of benefit. It certainly can do no harm. Gastric lavage is useful if fermentation of the gastric contents is present. He considers the employment of the *x*-rays to be of little use. Radium, locally applied, especially in malignant stricture of the œsophagus and in cancer of the stomach, has a decidedly beneficial effect.

Œsophageal Stricture Treated by Injections of Fibrolysin.—Weisselberg (*Münch. med. Woch.*, 1906, No. 33, p. 1623) reports an instance of stricture of the œsophagus following ingestion of a caustic. Following twenty injections of fibrolysin into the skin of the back, at two to three day intervals, the upper stricture permitted the passage of a good-sized sound. A second contraction was, however, demonstrated at the cardia; this would not allow the passage of the finest instruments. After thirty-nine injections this became patent to a small sound and finally complete cure was attained. No evil effects other than slight local pain, and on two occasions small abscesses, followed the injections.

Creosote Irrigations in Dysentery.— G. Zanardini (*La semaine médicale*, 1906, xxvi, 462) has treated successfully eleven instances of dysentery by irrigations of an emulsion of creosote prepared by adding to a quart of water, with which the yolk of an egg has been mixed, a dram of 10 per cent. creosote. Billet has also employed creosote irrigations in dysentery due to the Entamœba hystolytica of Schaudinn. He gives irrigations of from one pint to one quart of 1 to 2 per cent. creosote, which has been previously mixed with oil of sweet almonds, in water. Usually two enemas a day are prescribed. In a large majority of the patients thus treated, the pain is rapidly relieved and the blood and the fœtor disappear from the stools.

Vegetable Iron; the Therapeutic Employment of Rumex Crispus.—Gilbert and Lereboullet (*Les nouveaux remèdes*, 1906, xxii, 361) state that iron exists in plants in loose combination and in masked form. The former can be easily dissociated by the usual reagents, but the latter is resistant to these. Practically the therapeutic utilization of iron in masked combination is difficult on account of the small quantity which most vegetable substances contain. Amongst these Rumex crispus is an exception and is able to render to the organism a considerable amount of iron, if cultivated under certain conditions, fertilization with iron carbonate being the most important of these. By this procedure the roots may be cultivated so as to contain 0.447 per cent. of iron. The dosage of the pulverized root is from 7½ to 11 grains given in powders or capsules at meal-time. The best results are obtained from this drug in chlorosis and in the anemia of tuberculosis.

Milk as a Hemostatic.—P. Solt (*Therapeutische Monats.*, 1906, xx, 479) has employed milk as a hemostatic in internal hemorrhage with excellent results. The milk is given by rectal enema, either warm or cold and in quantity of one pint to one quart. Rarely is a larger amount necessary, but at times Solt has injected three quarts at a time. The patient is placed upon his side and the buttocks are held together so that the enema may be more easily retained. The milk is always well borne, but sometimes the patients complain of distention. The treatment is useful in all classes of internal hemorrhages and especially those due occurring as a result of genital disorder in the female. Solt has employed it in profuse menorrhagia, uterine hemorrhage following abortion or due to retained placenta, in hematemesis due to gastric ulcer, in hemoptyses, and in hematuria due to vesical cancer.

The Quinine Treatment of Cholera.—E. B. Fullerton (*Jour. Amer. Med. Assoc.*, 1906, xlvii, 1986) considers that quinine has always proved the best remedy in Asiatic cholera. The drug given in the form of the sulphate, in doses of 10 grains an hour, produces the best results. The bisulphate is more soluble and is more likely to be absorbed. For the purpose of disinfecting the intestinal tract the drug should not be given in solution, but should be taken moistened with only a little water. Only slight symptoms of cinchonism have been observed when the comparatively insoluble sulphate has been used. Larger doses than 10 grains have been given, but are likely to be followed by vomiting. The fear of markedly ill effects from the doses recommended may be set aside, as the lethal dose lies somewhere beyond one ounce of the drug, and, in addition, quinine is an antiemetic in cholera.

The Treatment of Arteriosclerosis.—Osborne (*Yale Medical Journal*, 1906, xiii, 115) states that in the prophylaxis of this condition muscular activity, nervous strain, and everything that tends to increase peripheral tension should be avoided. Large amounts of water or other liquids should not be taken, for they tend to overfill the bloodvessels. A modified, diminished, mixed diet, alcohol-free, if possible, without contractor stimulants, as caffeine, coffee and tea, spices or any drug, such as strychnine or digitalis, unless there is actual cardiac failure, will give the best results. Physical and mental rest are of great value; the moderate use of tobacco, after thirty years of age, may be of benefit in instances of hypertension; in excessive amount it will, however, precipitate cardiac insufficiency. If there is no kidney or liver involvement the vasodilatation resulting from a little alcohol at night may be of benefit, but the same results may be obtained from small doses of glyceryl nitrate ($\frac{1}{400}$ grain) three or four times a day. The artificial serum of Trunecek may be employed hypodermically. This consists of a combination of small amounts of sodium chloride, sodium sulphate, sodium carbonate, and potassium sulphate in distilled water. These substances may also be given by the mouth. Their efficiency is based on the theory that they introduce into the circulation a solvent which will dissolve the calcium phosphate which has been deposited in the bloodvessel walls, but the originator claims that all the disagreeable symptoms due to hypertension disappear under this treatment and that blood pressure is positively lowered.

The Intravenous Injection of Strophanthine.—Fraenkel (*Deut. med. Zeit.*, 1906, No. 62, p. 695) employs strophanthine intravenously in instances of cardiac insufficiency such as usually are benefited by digitalis. The dosage is from 12 to 15 minims of a 1 to 1000 solution, and the effects of the administration of this amount become evident within a short time; the pulse becomes fuller, the dyspnœa is relieved, and diuresis is established. The great advantage of the treatment is that all digestive disturbance is avoided, but it must be remembered that a second dose should not be given before the effect of the first has worn off, which would seem to show that strophanthine has a cumulative effect. Subcutaneous injections of strophanthine are very painful, but its intravenous administration is attended by no such untoward effect.

PEDIATRICS.

UNDER THE CHARGE OF

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Retarded Cerebral Development due to other than Cerebral Causes.—Ernest Laplace (*Penna. Med. Jour.*, January, 1907, p. 277) refers to a class of children whose heredity is not shrouded in suspicion and whose environment likewise is good, but who, nevertheless, are backward and are mentally and physically below par. They are slow in retaining mental impressions, they show little or no tendency to learn to talk, suffer with a variety of nervous phenomena, and have every evidence of imperfect nutrition; they are mouth breathers, and present exacerbations of temperature, which depress more or less the nervous system. With the exclusion of abnormality in the shape or development of the head, and of the history of a previous meningitis, the examination of these children reveals with wonderful frequency the presence of three abnormal conditions, namely, postnasal and pharyngeal adenoids, tongue-tie, and phimosis.

The pathological results of these deformities can be classified as follows: (1) From the adenoids, improper and insufficient aeration of the blood, resulting in insufficient combustion and imperfect general nutrition, and impairment of hearing resulting in sluggishness and lack of interest. (2) From the tongue-tie, a hampered condition of the tongue in its attempts to articulate. (3) From the phimosis, an attendant reflex irritation of the nervous system and the general depression which soon follows.

A number of such patients have come to Laplace's attention. Almost without exception boys present the combined defects mentioned. The condition has not been found proportionately as frequent among girls, inasmuch as the tongue-tie and adenoids would be the only causative elements. Surgical correction of these frequently associated defects, carried out in 82 cases under Laplace's care, has been followed by most encouraging results, and seems to confirm his suspicion that this strange coincidence of defects in the same patient often contributes an important factor in the retardation of mental progress.

Tetany Considered as an Intoxication by the Salts of Calcium.—W. Stoelzner (*Jahrb. f. Kinderheilk.*, 1906, xiii, 661) states that Finkelstein has called attention to the fact that frank or latent tetany can be influenced by diet. He has observed that with such patients the galvanic hyperexcitability of the nerves, as well as most of the other symptoms, are aggravated by the administration of cows' milk. On the contrary, after purgation and institution of a watery diet, the phenomena disappear completely in the course of twenty-four hours. Cereals prepared without milk act in the same way. But if after this amelioration cows' milk is given the symptoms reappear. In investigating the cause of this action of cows' milk Finkelstein has been able to establish the fact that casein, butter fat, sugar of milk, and even human milk, when added to cereals, are without action on the tetany. On the contrary, the serum of cows' milk acts exactly in the same fashion as whole milk—whence he has concluded that tetany is determined by a disturbance in the nutritive processes depending probably upon the assimilation of the phosphates or the calcium salts. This point has been made the subject of Stolzner's study of the galvanic excitability of the peripheral nerves in infants affected with tetany, before and after the administration of cows' milk or the calcium salts.

While the 12 cases reported by the author are not equally significant it seems conclusive that the administration of cows' milk or the calcium salts caused a manifest exaggeration of the excitability of the peripheral nerves, while this effect was wanting when, as a control, these infants received phosphate of sodium, lactate of iron, acetate of potassium, chloride of sodium, or magnesia.

The mechanism of this action of the calcium salts in producing or exaggerating the symptoms of tetany is thus explained by Stoelzner:

Tetany is exceptional with the breast-fed infant, but is almost always observed with infants nourished by cows' milk, which contains five times more calcium than human milk. The absorption and elimination of calcium salts being effected by the intestinal mucosa, it is evident that this organ is subjected to a superalimentation, which is aggravated if there are digestive disturbances, or if there is rachitis, in which the bones imperfectly utilize the calcium absorbed. It may, therefore, result with infants nourished with cows' milk, whether or not they become dyspeptic or rachitic, that the intestine can become functionally insufficient in eliminating the calcium salts and thus lead to a retention manifesting itself clinically by tetany. It remains to be proved whether in such cases the blood and organs are surcharged with calcium salts, a point upon which so far no researches have been made.

A Vegetable Broth Useful in Gastro-enteritis.—Bailey (*Gaz. hebd. des Sci. méd.*, June 2, 1906) gives a recipe for a broth which may be used with advantage when it is necessary to interdict milk as a food in case of milk infection: Carrots, 65 grams; potatoes, 5 grams; turnips, 25 grams; dry peas or dry haricot beans, 25 grams, for each liter of water. Cook well and strain, adding 5 grams of salt per liter.

The Alimentary Regimen of Measles.—E. Ramus (*Régime alimentaire à instituer dans la rougeole, Thèse de Paris*, 1906) makes some valuable suggestions as to diet of children suffering from measles, based upon a study of urinary elimination and the weight. Dividing the duration of the disease into periods of four days each, counting from the day of eruption, Ramus has observed that the quantity of urine passes through three phases: First, a phase of oliguria corresponding to the first period of the disease; a second phase of polyuria coincident with the second period; and a third, covering the last two periods, in which the quantity increases, diminishes, or remains stationary. This curve, which is more or less accentuated according to the gravity of the disease, is independent of the regimen adopted.

The elimination of urea shows notable modifications during the course of the disease. In the first period it is low; in the second it increases progressively, attaining a maximum from the ninth to twelfth day, after which it reverts to the normal. The administration of an albuminoid diet does not appear to increase the proportion of urea during the first period; it does augment urea elimination, however, during the subsequent periods; but it is impossible to establish an absolute parallelism between the curves of urea, the urine, and the course of the temperature.

The elimination of chlorides is not influenced by measles, and neither retention nor crisis is observed, as exists in other infectious diseases: the elimination is equal to the ingestion. No connection can be established between the excretion of chlorides, on the one hand, and that of urea, on the other.

The weight of the body in the course of measles undergoes a more or less considerable decline from the beginning of the incubation. In the following periods it remains stationary, to increase again without always attaining the original figure before complete recovery.

The chlorides seem to have a manifest influence upon the weight; if ingested from the beginning of the disease they retard the loss of weight, often so considerable during the first period. This attenuation cannot be attributed to fixation of water in the organism, since there is no retention of chlorides during the course of the disease. The role of the chlorides seems to be a biological one, consisting in a favorable modification of the phenomena of cellular nutrition.

The alimentary regimen proposed by Ramus is as follows: In the first four days of the disease it is useless to give albuminoid foods, since the child does not eliminate more urea under milk feeding than under barley-water. Give therefore water in the form of warm infusion of potherbs or mallow, barley-water or hot lemonade. To prevent too great a loss of weight, add to this a certain amount of sodium chloride, a little greater than that which would be taken in milk, about 5 grams for a child of from four to ten years. In the second period, after the fall of temperature, albuminoid foods may be resumed, since there is a sensible diminution of urea in children who do not receive albumin. Therefore, according to the appetite of the child and the condition of the alimentary tract, without recourse to an intermediate diet of milk exclusively, one can order without danger a diet suitable to the age of the child: broths, eggs, meat-juice, rice, butter, etc., watching always the condition of the stools, and reducing the diet if diarrhœa appears. In this way many of the complications resulting from the pronounced exhaustion of the disease may be avoided.

OBSTETRICS.

UNDER THE CHARGE OF

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Eclampsia.—Esch (*Ztschr. f. Geb. u. Gyn.*, 1906, lviii, Heft 1) contributes a paper upon this subject from studies made in Olshausen's clinic in Berlin. Between the years 1900 and 1905, in 6657 cases of labor, eclampsia occurred in 7.45 per cent. This is a high rate for continental clinics, the nearest approach to this in Germany being that of the Leipsig clinic, with 2.07 per cent. Of these, 78.3 per cent. were primiparæ, 21.7 multiparæ. Among the primiparæ were 14.8 per cent. older than 28. Nine of these patients had eclampsia in a previous pregnancy or parturition. Cases are on record in which the mother has had eclampsia in three successive pregnancies. Esch reports 3 patients who had eclampsia comparatively early in pregnancy, from which they recovered and subsequently had eclampsia at the time of the birth of the child. He adds also 9 other cases in which eclampsia occurred without the interruption of pregnancy, which subsequently went on to term. Among the symptoms of threatened eclampsia, the most significant was headache. Pain in the stomach and vomiting occurred just before or during convulsions. Disturbance of the special senses, especially sight, was frequently observed. An aura was not detected. The results of Esch's studies, with those of Olshausen and others, show clearly that eclampsia may occur after labor. The percentages are: Esch, 15.7 per cent.; Olshausen, 14 per cent.; Goedecke, 18.2 per cent.; Knapp, 14.6 per cent.; Goldberg, 1[illegible].88 per cent.; Winckel, 17 per cent.; Löhlein, 23 per cent.; Braun, 24 per cent.; Schauta, 24.6 per cent.; Büttner, 32 per cent.; Wirz, 19.7 per cent.; Hammerschlag, 19 per cent.; and Bidder, 29 per cent.

Eclampsia may develop as late as two days after labor. In one case reported by Esch, convulsions occurred nine days after the birth of the child. The longest interval reported between childbirth and eclamptic convulsions is eight weeks.

On admission, most of these patients were comatose, with labored respiration, rapid pulse, frequently with high tension, sometimes small and irregular. The patients were exceedingly restless and more or less unconscious. A moderate degree of fever was often present. Œdema was observed in the majority of cases in some portion of the body. In a considerable portion of cases the urine showed serum albumin. The specific gravity varied from 1010 to 1018, the urine was more highly colored than normal, and occasionally dark or reddish brown. The quantity was lessened. As a rule, immediately after labor the quantity increased or the albumin lessened. Debris from the kidneys was present in a large percentage of cases.

The convulsions were characteristic of eclampsia or epilepsy, and lasted from three-quarters of a minute to two or three minutes. The longest duration was twenty-two minutes. The number of convulsions varied greatly. One patient had 146 convulsions; 2 patients had none, a condition considered exceedingly rare. In the patient who had 146, 5 occurred before labor, and 141 after labor. On the fourth day after the birth of the child the patient had 65 convulsions. While it is true that in many cases convulsions cease after the birth of the child, this is not invariably the case. It is probably safe to say that convulsions will cease in 49 per cent. of cases after the uterus is empty. Forceps was used in 141 cases in the ordinary manner; in one case upon the after-coming head, and thirty-three applications were made. The number of operations in all was 267, which gives a percentage of operative interference of 77.6 per cent. Of these, 21 were performed upon patients in whom the vaginal portion of the cervix was still present and undilated.

Of the 496 patients, 106 died, or 21.4 per cent. The greatest mortality occurred in March and in November. Occasionally groups of cases were seen in which the mortality was enormously in excess, as during a few days in the early winter of 1905 and 1906, when 4 of 5 eclamptic patients died; 6 of these patients died before labor, and one died as late as on the nineteenth day after labor; 91 of these women died as the immediate result of eclampsia; of the others, 16 died from complications. Autopsy was obtained in 70 of these cases; 4 of these had septic infection, but in none of these could the infection be traced to operation.

There were 31 patients on whom the Bossi dilator was used; of these 14 died; 12 of these cases came to autopsy. In 8 of them the pathologists reported no laceration of the cervix; in 3, slight laceration upon one side; in 1, an extensive laceration. One of these patients died of septic infection, the others of eclampsia. It is thought to be remarkable that so little septic mortality developed in cases in which the cervix was so little dilated, and the Bossi dilator was employed. Attention has been called to the dangers of the use of anesthetics in these cases, and especially prolonged narcosis, which favors the development of pulmonary complications. The length of time necessary for dilating the cervix varied from five to sixty minutes. It could scarcely be proved that the length of time occupied in dilatation had a direct influence on the mortality. In puerperal eclampsia the mortality was 15.4 per cent. When the convulsions did not develop until fourteen hours, or longer, after the birth of the child there was no mortality.

The number of convulsions had a direct relation with the mortality in the majority of cases. In some, however, death occurred after but one or two convulsions, and this is ascribed to the uncommon virulence of the poison.

High temperature accompanied a high rate of mortality, and is considered a most unfavorable symptom. The quantity of albumin present in the urine was of no importance whatever in basing a prognosis. When casts were very abundant, indicating a rapid disintegration of the kidney, the prognosis was bad. When the urine was highly discolored, the cases were also grave.

As regards the determination of the prognosis, it is stated that this is never possible with exactness. When the patient is not deeply unconscious, breathes easily, and has a good pulse, the symptoms are comparatively favorable. In cases in which convulsions seem to be excited by the pressure of the fetal head at birth, or by the taking of stitches, the prognosis is also better. When coma is deep, the pulse small and rapid, and the temperature rises, the prognosis is bad. Especially unfavorable are those subjects who pass into deep coma after the first convulsion. Icterus, hemoglobinuria, scanty secretion of urine, and anuria are all unfavorable. Those cases are most desperate which are seen by the physician after they have long been in convulsions, and are excessively weakened. Cases of primiparæ above the average age, and patients having great quantities of albumin, with abundant casts, are also unfavorable. If a patient improves after the birth of the child, such improvement is most promising. When this is absent, the prognosis is grave. An especially significant symptom is headache, which should grow very much less, or disappear, after the birth of the child. Especially dangerous are puerperal complications in the lungs, such as inspiration pneumonia or bronchopneumonia. The mortality of eclampsia is less in cities than in the country, because in cities patients obtain more prompt and efficient treatment.

The chance for the child is less favorable than for the mother. Olshausen believes that few children survive twelve or fifteen convulsions. Glockner observed that when more than ten convulsions occurred before the birth of the child, that but 17 per cent. of the children were born living. Narcotics given to the mothers affect the children unfavorably. When large doses of opium are given, the children are born narcotized and require active treatment for resuscitation. The mortality among children in these cases was 22.7 per cent. Others give a percentage ranging from 28.44 per cent. When spontaneous birth occurred the mortality was 15.7 per cent. When operation was necessary, it rose to 31.9 per cent. The mortality of 22.7 per cent. is unusually low, and is ascribed to the active treatment employed. Other observers give this mortality as ranging from 31 to 49 per cent.

It is remarkable that so few children born of eclamptic mothers have convulsions. In some cases the child's eclampsia occurs during its first bath. The children of eclamptic mothers should be guarded against irritation or excitement likely to induce convulsions.

As regards treatment, statistics are quoted which seem to show that immediate delivery gives the best results. In Esch's experience, rapid delivery by Bossi's dilator, or Dührssen's vaginal section, increased the mortality 4.5 per cent. Rapid delivery is not a panacea for eclampsia It is true that convulsions cease in the greater majority of cases after the birth of the child, but this does not prove that recovery will take place.

Dividing his cases into three periods, Esch tried during the first period incisions in the cervix, varying in length, followed by delivery. The mortality rate was 28.9 per cent. During the second period, incisions were made less frequently, and Bossi's dilator was introduced. The mortality was then 27.87 per cent. During the third period, in addition to other methods of treatment, vaginal Cesarean section was performed, when the mortality fell to 19.8 per cent.

When a comparison is made between different classes of cases, and the statistics of other operators are taken into consideration, it is found that the emptying of the uterus must be chosen with regard to the condition of each individual patient. When delivery is selected, the use of forceps is usually made available; occasionally version with extraction; when the child is already dead, perforation and the use of the cranioclast.

When non-operative treatment is considered, morphine, chloral, bromide of potassium, are used, in accordance with individual indications. Narcosis is employed for all interference. Stimulants are given to excite the action of the heart. During the puerperal period the diet is milk, and acetate of potassium is administered as a diuretic. During operative delivery, ergot is given hypodermically. When Cesarean section, abdominal or vaginal, is made the patient receives an injection of ergot fifteen minutes before the operation. In all cases, the patient is disturbed as little as possible, and a bright light is carefully excluded from the room. When respiration is labored, the foot of the bed is raised and the patient's head turned to one side to permit the free escape of secretions from the mouth. The mouth is frequently cleansed with sterile gauze. Wet compresses and packs are used to allay excitement, although they are thought to depress the patient greatly. Stimulating injections are frequently given before a pack. No diaphoretic treatment is carried out, as it is thought to be useless or harmful.

Esch did not try spinal puncture or spinal injections of scopolamine and cocaine. In plethoric cases, bleeding followed by subcutaneous injections of salt solution proved useful.

The ideal treatment is prophylaxis, and especially the strict regulation of diet during pregnancy. In making incisions in the cervix, the anterior and posterior portions are selected. Usually two incisions suffice.

Hemorrhage and laceration in these incisions was not observed. They were not closed by stitches after the birth. In employing the Bossi method, the instrument devised by Frommer is employed. When vaginal Cesarean section is done, anterior incisions are usually made. When this does not suffice, then incisions are made posteriorly. The child is delivered by version and extraction. In primiparæ, deep, lateral incisions are sometimes made through the labia.

GYNECOLOGY.

UNDER THE CHARGE OF

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Origin of Embryomas.—Freund (*Zentralbl. f. Gyn.*, 1906, No. 44) raises the question whether embryomas may not develop from aberrant blastomeres, as well as in accordance with Bonnet's theory of congenital inclusions. He believes that the clinical symptoms of this class of neoplasms nearly always point to a previous pregnancy, the fetal inclusions being developed under the influence of puerperal changes. It is conceivable that besides the fruitful ovum a second may be fecundated at the same time, and that this perishes except a single blastomere.

This theory is supported by the fact that in some cases the usual changes in the uterus and breasts and the presence of the corpus luteum of pregnancy have been observed in connection with an ovarian embryoma. Certain striking changes which take place in the walls of dermoid cysts, not observed in other varieties of cystoma, may be regarded as a peculiar reaction of the ovarian remains to the implantation of a blastomere, like the dilatation and increased growth of the lymphatics seen in the pregnant uterus. The fecundation of an ovum in the atrophied stromal remnants of a cystoma may also result in the development of an isolated blastomere and thus lead to the formation of an embryoma.

Removal of the Ovaries in Myomotomy.—Rochard (*Bull. gén. thér.; Zentralbl. f. Gyn.*, 1906, No. 43) pleads for extirpation of the adnexa in every case of hysteromyomectomy, since the ovaries may subsequently become inflamed and cystic, or serious intraperitoneal hemorrhage may result from ruptured follicles (?). He thinks that the arguments advanced for preservation of the ovaries after removal of the uterus are not convincing, the reflex disturbances being unimportant and the theory of internal secretion fanciful. (It is evident that this paper is based on limited clinical experience, else the writer would not indulge in dogmatic statements, opposed to that of the majority of abdominal surgeons.—H. C. C.)

Operations for Recurrent Carcinoma.—Franz (*Zentralbl. f. Gyn.*, 1906, No. 44) reports 16 such operations for recurrent disease following hysterectomy; 9 patients were operated upon once, 2 twice, and 1 three times. In 9 there were no complications; in 2 the base of the bladder and portions of both ureters were resected, and in 1 a ureterovaginal fistula was cured; in 2 resection of the large intestine was necessary, while one kidney was extirpated in two instances.

One patient died after removal of a pyonephrotic kidney and one of embolus two weeks after operation; a third succumbed from extension of the disease six months later.

Franz believes that whenever possible the patient should have the benefit of surgical treatment; though life is thus prolonged and pain relieved, a permanent cure can rarely be hoped for. The indications for operation are limitation of the growth and a certain degree of mobility, showing that it is not firmly adherent to the pelvic wall. Care should be taken to avoid injury to the rectum and bladder, and it is indispensable that the kidneys should be in a healthy condition.

Peritoneal Adhesions.—Pankow (*Zentralbl. f. Gyn.*, 1906, No. 44) from experiments on animals arrives at the following conclusions: No adhesions result from superficial lesions of the visceral or parietal peritoneum, even when cauterized. In 50 per cent. of the cases in which the parietal peritoneum was roughly scraped, or punctured at several points, adhesions occurred. Slight adhesions resulted when the cautery, or alcohol, was applied to check oozing. In artificial staphylococcic infection it was found that the power of resistance was greatly lessened when the peritoneum was injured, especially if the raw surfaces were touched with the cautery or with alcohol.

Ovarian Pregnancy.—Schickele (*Zentralbl. f. Gyn.*, 1906, No. 44) adds a third case to those reported by Franz and Van Tussenbroek. In one there was no question that the fecundated ovum lay within a ruptured follicle; in another, the remains of the ovum besides the fresh corpus luteum were free in the tissues, so that the fetal cells were in direct contact with the maternal connective-tissue cells. In Schickele's case the ovum was in close contact with the corpus luteum and had caused marked distention of its wall. In both of the last two cases the ovum was imprisoned within the stroma (epovarian implantation), in contrast to the intrafollicular situation.

Structure and Changes in Intraligamentary Tumors.—Schäffer (*Zentralbl. f. Gyn.*, 1906, No. 44) from a study of serial sections of an intraligamentary fibroid found that the abdominal portion of the neoplasm was cystic while the intrapelvic was fibromyomatous, receiving its vascular supply from the anterior fold of the base of the broad ligament, and having no connection with the uterus. Myxoid degeneration and pseudocysts were noted. In the capsule of the cystic portion of the tumor were large hard nodules resembling organized tissue, quite vascular. The arteries were greatly dilated, their walls showing hyaline changes, and terminated in capillaries which penetrated the myxomatous and œdematous tissue, the degenerative processes being most marked at a distance from the hilum, or point of entrance, of the nutritive vessels.

Vapocauterization of the Uterus.—Baisch (*Frauenärzt*, November 16, 1906, p. 504) believes that the therapeutic action of vapocauterization depends mainly on the condition of the endometrium. Since we cannot know beforehand the pathological lesion, we cannot affirm what will be the exact result. He is opposed to its use in young women, on account of the result on menstruation and conception. In those who have reached the climacteric other changes may be present, such as arteriosclerosis, so that vapocauterization may have no more effect than simple curettement.

Previous curettement is advised, from five to seven days being allowed to elapse before vapocauterization is used, an anesthetic being then unnecessary. It is absolutely contra-indicated in cases of uterine fibroid, as well as in gonorrhœal and septic endometritis.

He would limit the employment of this agent to cases of uncomplicated preclimacteric hemorrhage.

Bier's Method of Intra-uterine Treatment.—Zuran (*Zeit. f. Gyn.*, 1906, No. 28, p. 785) finds that the introduction of Bier's catheter is free from danger, provided that strict asepsis is observed. It relieves dysmenorrhœa, diminishes the secretion in cases of chronic endometritis, and exerts a curative action on the diseased endometrium, which extends to the submucosa. This method of treatment may be used in dispensary practice and does not interfere with the patient's occupation.

Thrombosis of the Vessels in Ectopic Gestation.—Fellner (*Archiv f. Gyn.*, 1906, Band lxxiv, Heft 3) describes the anatomical conditions in three cases of early tubal pregnancy. Numerous arteries in the neighborhood of the intervillous space were seen filled with round and spindle cells, which Fellner believed to be not of ectodermal origin, but derived from the subendothelial connective tissue. He regarded the condition as an "autothrombosis" in the arteries, and not as an escape of Langhans' cells into the veins, as stated by Veit and others.

He suggests that the invasion of the vessels of a myomatous uterus by villi, described by Poten, is really an arterial autothrombosis—hence the frequent occurrence of abortion in such uteri. Moreover cases of so-called chorion epithelioma may be explained in the same way.

Malignant Degeneration of Uterine Fibroids.—Winter (*Zeitschrift f. Geb. u. Gyn.*, 1906, Band lvii, Heft 1, p. 8) remarks on the extreme rarity of cancerous degeneration of fibromyomas of the uterus. Among 2331 cases of uterine cancer the body was affected in 151, myomas being present in 80. Hofmein, Fehling, and Winter together note 1.2 per cent. of cases of cancer of the corpus uteri with coexisting fibroids. In 1270 cases of cancer ot the cervix uteri there were 2 per cent. of fibroids, hence the fear that the stump may become cancerous after supravaginal amputation is practically groundless. When the symptoms of cancer appear in connection with fibroid uteri (especially atypical hemorrhages and discharges after the menopause) the presence of adenocarcinoma should be suspected and total extirpation is preferable.

The question of sarcomatous degeneration is different. Among 1734 cases of fibromyoma, 46 underwent sarcomatous degeneration (2.7 per cent.). This change is most likely to occur in the submucous variety (about 9 per cent.).

In conclusion, Winter lays little stress upon the danger of malignant degeneration of fibroids as an indication for operation, though he admits the frequency of cystic, suppurative, and other benign changes.

Results of Radical Abdominal Hysterectomy for Uterine Cancers.—Brunet (*Archiv f. Gyn.*, 1906, Band lxxviii, p. 632) reports 251 cases in which the uterus, parametric tissues, and lymph nodes were examined microscopically after removal. He notes that the average operability is only 30 per cent., the average mortality being 15 per cent. He still advises radical removal of the diseased organs and tissues, even the upper third of the vagina, as well as the broad ligaments and glands. No comments are made with regard to the permanency of the "cure."

Premonitory Bleeding in Ectopic Gestation—Schickele (*Zentralbl. f. Gyn.*, 1906, No. 44, p. 1227) calls attention to the fact that slight hemorrhages from an ectopic sac preceding the final intraperitoneal rupture may easily lead the observer to infer that the case is one of simple tubal abortion. It is well known to every operator that such premonitory hemorrhages often occur, as shown not only by the finding of small openings in a sac removed between the third and fifth months, but by the presence in the cavity of blood clots of different ages. (This interesting paper only serves to emphasize the importance of early operation, or at least of diagnostic vaginal section, at the first appearance of suspicious symptoms, whether associated with a tumor or not. No case of ectopic gestation should be allowed to progress as far as the third month.—H. C. C.)

DERMATOLOGY.

UNDER THE CHARGE OF

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A Case of Dermatomycosis Caused by Microsporon Canis.—NICOLAS and LACOMME (*Annales de Dermatologie et de Syphiligraphie*, 1906, No. 4) report the following case: A man, aged thirty-seven years, complained of generalized itching, most marked on the anterior and lateral surfaces of the thorax. No evidences of scabies or pediculosis were found, but on the anterior surface of the chest, about the middle of the left clavicle, and in the left axilla were two erythematosquamous patches about the size of a two-franc piece. Microscopic examination of scales from these plaques revealed the presence of numerous mycelial threads. Upon interrogating the patient it was learned that he was in the habit of playing with a dog which presented typical lesions of tinea tonsurans. By means of culture and inoculation experiments, the identity of the fungus found on the patient and that obtained from the dog was established, the fungus being identified as Microsporon canis. The disease rapidly disappeared after the use of sulphur baths accompanied by frictions with black soap, and painting with tincture of iodine.

A Case of Folliculitis Cutis Gonorrhoica.—CRONQUIST (*Archiv f. Dermatologie und Syphilis*, Band lxxx, Heft 1) reports the following unusual case of gonorrhœal infection of the skin: A young man, aged twenty-one years, suffering from a gonorrhœa of one week's duration, with the usual symptoms, an abundance of gonococci being found in the urethral discharge, presented a pea-sized, markedly inflammatory nodule situated a short distance below the umbilicus. Upon removal of the crust which covered its top a drop of greenish pus welled out in which gonococci were demonstrated. Microscopic examination of the lesion showed that the process was a folliculitis and perifolliculitis and its gonorrhœal nature was proved by the reaction of the pus with the Gram stain. (Cases such as above, and a few others reported by other observers, prove conclusively that the gonococcus may give rise to cutaneous disease.—L. A. D.)

Two New Cases of Porokeratosis.—MIBELLI (*Brit. Jour. of Dermatology*, May, 1906, p. 197) states that about forty cases of this disease have been published. Two new cases are recorded, both occurring in field laborers, one young, the other old, the regions affected in both being the face, hands, legs, and genital region. The chief manifestation consists of a hyperkeratosis together with the characteristic feature of the depressed furrow of the lesion. The process is regarded by Mibelli as being really a porokeratosis rather than a hyperkeratosis. Later atrophy takes place in the diseased areas, which are thus found when the sebaceous glands and the hair follicles are equally destroyed. In addition to these changes there is a slight infiltration of the corium with mononuclear leukocytes, together with disappearance of the elastic network and a thinning of the callogen bundles. This deeper inflammation is probably the sequel of the hyperkeratosis rather than *vice versa*. The histological facts may be summed up by stating that the disease is a hyperkeratosis with porokeratosis, followed by inflammation of the corium and, finally, atrophy, the hyperkeratosis electing the glandular and follicular apertures, with special reference to the sweat ducts.

Pathology and Therapy of Yaws.—ALDO CASTELLANI (*Jour. of Trop. Med.*, January 1, 1906) concludes that both the lesions and the treatment go to show that yaws (known also as frambesia, parangi, pian, etc.) is distinct from syphilis. The early lesions of yaws show a spirillum, for which he proposes the name Spirochæta pertenius, but this spirocheta does not prove that the disease is identical with syphilis. Large doses of potassium iodide he found useful in yaws, and much more effective than mercury. Some cases recover without treatment.

Mucous Membrane Lesions in Lupus Erythematosus.—THOMAS SMITH (*Brit. Jour. of Dermat.*, February, 1906) has made a study of fifty-six consecutive cases of lupus erythematosus and has found that sixteen had some affection of the mucous membrane, giving a percentage of 28, which is greater than the prevalent idea. The majority of the patients were unaware of the existence of any lesions in the mouth. All had been patients at the London Hospital recently, the observations having been corroborated by Dr. Sequeira in nearly every case. In no instance of the noted cases was the tongue affected. The mucous membrane lesions were more frequently encountered in cases of the disseminated type. The locality affected was generally the inner surface of the cheeks. The lesions do not materially affect the course of the disease of the skin and do not appear to demand any special local treatment.

PATHOLOGY AND BACTERIOLOGY.

UNDER THE CHARGE OF

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Studies in Beriberi.—HERZOG (*Philippine Jour. of Science*, 1906, i, 169) in two papers discusses beriberi, with special reference to the very obscure question of its etiology. In the first paper the author gives an account of his work in the Japanese Army Hospital during the late war. He points out that although the very efficient sanitary arrangements in the field were successful in combating typhoid fever, dysentery, scorbutus, and typhus fever, beriberi, or kakke, as the Japanese call it, was not preventable or apparently controllable in the actively employed army. Herzog gives an account of the work of Okata and Kobubo, who have isolated a coccus from numbers of cases of beriberi. The blood of 129 cases was examined, both microscopically and with cultures, and sixty-five times both methods were successful. In 35 cases the organism was not found by either method, and in 11 the microscope alone revealed it, while in 19 cases this method was negative, but successful cultures were made. The organism usually occurred as a diplococcus, but at times as a staphylococcus. In smears it was seen both within and outside of leukocytes. It never showed a capsule or motility. It grows readily on the usual culture media. All animal inoculations were unsuccessful. The coccus was isolated by these Japanese authors twenty-five times from the urine of 34 patients, and obtained fifteen times from the feces of 45 patients. Herzog obtained it eight times in 30 to 40 attempts at isolation from the urine of patients suffering with beriberi.

The second paper is a more general account of the disease, and is based upon the study of beriberi as seen in the Philippine Islands. The various etiological factors for the disease, which have been more or less hypothetically advanced, are discussed. In order to test the idea of Donald Ross that the disease is caused by arsenical poisoning, the hair of the patients was analysed for arsenic, with negative results. Herzog has undertaken an extensive study of the Okata-Kobubo coccus, and finds that the strains of the organism which he used corresponded culturally almost entirely to the characteristics reported by the Japanese observers. His results with animal inoculations are entirely negative, although quite extensive. Six series of monkeys, guinea-pigs, rabbits, and rats were used, and observed over a long time. Blood cultures were made from 40 patients with better technique than that employed by the Japanese, and all were sterile except in 6 cases, in which the cultures were apparently contaminated. In one fatal case the Okata-Kobubo coccus was isolated from the much enlarged spleen, and with this organism all animal experiments were negative. Herzog was able to produce a very low grade of agglutinating serum by repeated injections of the organism.

He concludes that beriberi is neither primarily a nutritional disturbance nor a simple intoxication like lead, alcohol, arsenic, or similar intoxications accompanied with multiple peripheral neuritis, but is an infectious disease. His blood examinations and animal experiments indicate very strongly that none of the claims brought forward for the discovery of a specific micro-organism for the disease can be looked upon as substantiated. Herzog advances the hypothesis that the disease is due to some organism which gains entrance to the body and then produces an extracellular toxin, which may be compared to diphtheria and tetanus toxin, and which, by an accumulative action, gives rise to the well-characterized anatomical lesions of beriberi.

Researches on Immunity to Carcinoma in Mice.—Ehrlich has found that by injecting mice with spontaneous or transplanted avirulent tumors an immunity can be obtained against virulent tumors. Repeated injections of avirulent material increase the immunity. Not only is there an immunity for the same type of carcinoma but for other strains, and if the immunity for carcinoma is carried to its maximum the mouse is likewise protected against inoculations of sarcoma. The reverse is also true; and in one case a mouse immunized against sarcoma showed partial immunity to inoculations of a transplantable chondroma. This question of "pan-immunity" has been tested still farther by Borrel, who failed in attempts to immunize mice against carcinoma by injections of the livers of mice, and by Bashford, who claims to have produced immunity against the Jensen tumors by injections of the blood of mice.

Schöne (*Münch. med. Woch.*, 1906, liii, 2517), working in Ehrlich's laboratory, has repeated these experiments. He has tried to determine if mice injected with various cells and tissues, other than the tumors of mice, develop any immunity against mouse tumors. For this purpose the embryos of mice, the livers and testicles of mice, chicken embryos and carcinoma of the human mammæ were used. Schöne found that repeated intraperitoneal injections of mice embryos seemed to protect the animals against subsequent inoculations of transplanted carcinoma. In a series of 18 inoculated mice 12 (or 66.67 per cent.) did not develop carcinoma four weeks after the inoculation, while in the 18 controls only 3 (or 16.67 per cent.) proved refractory. With the liver, testicles, chicken embryos, and mammary carcinoma the results were not striking; and though mice treated with these tissues seemed to be more resistant to the inoculations of carcinoma than the controls, the author thinks that the number of his experiments is too small for him to draw definite conclusions. A second series of mice which received repeated injections of the same tissues, except for the mammary carcinoma, showed no immunity against inoculations of a virulent sarcoma.

Active Immunization of Man against Typhoid.—FRIEDBERGER and MORESCHI (*Deut. med. Woch.*, 1906, xxxii, 1986) prepare an exceedingly powerful vaccine by drying mass agar cultures in an exsiccator, and heating for two hours at 120° to 150° C. At this temperature the bacilli are killed, but the antigens are not destroyed. To test the value of the vaccine fourteen young men, who, of course, had not had typhoid, were inoculated. Injections were made intravenously. Doses of $\frac{1}{50}$ oese produced somewhat alarming symptoms, while much smaller amounts gave rise to high fever and chills. Indeed, the vaccine proved so active that 0.00078 milligram was sufficient to give rise to definite symptoms. After injections of the vaccine the serum of the individuals agglutinated the typhoid bacillus in high dilutions and showed a marked increase over the normal in bacteriolysis.

Typhoid Bacillus Carrier.—The name "bacillus carrier" was given by FORSTER and KAYSER (*Münch. med. Woch.*, 1905, No. 31) to persons, who weeks or years after an attack of typhoid fever, eliminated typhoid bacilli, as proved by culture, in their stools. They described several such cases and suggested that the gall-bladder acted as a reservoir for the typhoid bacilli. LEVY and KAYSER (*Münch. med. Woch.*, 1906, liii, 2434) report the case of a woman who, in 1903, suffered an attack of typhoid fever. During the winter of 1905 typhoid bacilli were found on ten different occasions in her stools. In October, 1906, she became ill and died of pneumonia. At autopsy the gall-bladder contained a concretion. Enormous numbers of typhoid bacilli were found in the bile, and were cultivated from the centre of the stone. Typhoid bacilli were also found in the spleen and liver. Levy and Kayser conclude that the gall-bladder is the seat of vegetation for typhoid bacilli in the "chronischer Bacillenträger," and point out, moreover, that from the gall-bladder an autoreinfection may take place.

Observations on the So-called Abnormal Fibrous Bands of the Heart.—Tawara (*Ziegler's Beitr. z. path. Anat.*, 1906, xxxix, 563) has studied these fibrous bands which are not infrequently found in the ventricles, and either cross the cavities or run as isolated bands along the walls, and finds that in their direction they follow the course of the fibers of the atrio-ventricular bundle of muscle fibers. According to Tawara they represent isolated bundles of fibers forming a continuation of the muscular fibers of the main bundle. Magnus-Alsleben (*Cent. f. Path. und Path. Anat.*, 1906, xvii, 897) has been able to demonstrate that these anomalous fibrous bands which he has examined in ten different hearts contain muscle fibers, sometimes separated or surrounded by connective tissue. The muscle fibers are characterized by the presence of much sarcoplasm. This, as Tawara has stated, together with scanty development of fibrils is typical of the muscle fibers which form the continuation of the atrio-ventricular bundle beneath the endocardium of the ventricles.

Agglutinins and Specific Immune Bodies in Gonococcic Serum.—Vannod (*Deut. med. Woch.*, 1906, xxxii, 1984) has prepared a serum by inoculating rabbits with the nucleoproteid of gonococci which is highly agglutinative for the gonococcus. Though the gonococcic serum does not agglutinate staphylococci and streptococci it does agglutinate meningococci. A meningococcic serum agglutinates correspondingly the gonococcus. The gonococcic serum, however, contains antibodies, which are "bound" by the gonococcus, as tested by the method of complement deviation of Wasserman and Bruck; whereas the serum does not contain bodies that are bound by the meningococcus.

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THE
AMERICAN JOURNAL
OF THE MEDICAL SCIENCES.

MAY, 1907.

ORIGINAL ARTICLES.

THE DIAGNOSIS AND TREATMENT OF CARDIAC DEGENERATION APART FROM VALVULAR DISEASE.

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Clinically, cases of myocardial degeneration may be divided into two groups: (1) Those in which diagnosis is comparatively easy and certain and rests on good and sufficient data, which will be elaborated later on, and (2) those in which diagnosis is by exclusion or inference, such data as exist being few but often quite significant. In the first group symptoms of cardiac incompetence may or may not be present, but in this discussion they are presupposed. In the second group they are wanting or obscured by symptoms referable to structures other than the heart.

Group I. Age is highly important, for, although the myocardium may be comparatively healthy in exceptional instances up to advanced age, its degeneration grows progressively more probable with every year past middle life, and may be taken for granted when senility has been reached. Of importance also is a history of syphilis, chronic alcoholism, certain acute infections, as influenza, cholelithiasis, gout, lead or phosphorus poisoning, or any other of the agencies generally recognized to exert a malign influence upon the nutrition of the heart or arteries.

Symptoms of cardiac incompetence may possess great diagnostic value in connection with age, but when occurring in persons nearing forty years they must be considered suggestive rather than conclusive. In the latter event, due attention must be given to the history for the purpose of excluding the possibility of acute heart strain; and every endeavor should be made to detect some of the objective data soon to be mentioned.

Angina pectoris developing after the fiftieth year, and especially in connection with signs of cardiac hypertrophy or dilatation, suggests strongly coronary or aortic sclerosis, and hence degeneration of the myocardium. Precordial pain of a dull, oppressive character, coming on during exertion and usually associated with arterial hypertension, points to abnormal intracardiac blood pressure and chronic myocarditis. This is particularly true if the individual is in the middle period of life and if intercostal neuralgia can be excluded.

Breathlessness upon exertion in an individual past middle age, if not dependent upon emphysema or obesity, and many times even then, points to myocardial inadequacy; and if the dyspnœa displays the features of cardiac asthma coming on at night, there can be little if any doubt of degeneration of the heart muscle.

Vertigo is a very variable and equivocal symptom, but when associated with hypertension of the pulse may give early evidence of myocardial inadequacy; and cardiac insufficiency developing insidiously at or past the forty-fifth year should always arouse a suspicion of myocardial degeneration.

Another symptom, which in a suspected case always fills me with apprehension, is a peculiar combination of nervousness, anxiety, and air-hunger, the last being more subjective than objective perhaps. This syndrome is seen in connection with signs of cardiac dilatation, but to my mind indicates serious nutritional disturbance of the myocardium, and in three instances was soon followed by sudden death.

The objective data which warrant a diagnosis of cardiac degeneration are well known and need no elaboration. Signs of arteriosclerosis are generally held to be very significant, but may be pronounced in the arteries of the extremities without serious involvement of the aorta or coronary arteries. When, on the contrary, signs and symptoms denote extensive sclerosis of these latter vessels, serious degeneration of the heart muscle may be diagnosticated, and symptoms of incompetence are rarely wanting. These signs are moderate dulness in the right first interspace close to the sternum, a ringing and metallic or clanging quality of the aortic second tone, with or without a systolic murmur in the same area, and exaggerated pulsation of the subclavian arteries which arch high upward above the middle third of the clavicles. Under such circumstances the arch of the aorta is very likely to be thinned and dilated.

Bradycardia may occasionally possess diagnostic significance, but unless showing the Adams-Stokes peculiarity is not so important as is persistent acceleration of the pulse. Tachycardia is an especially valuable sign of myocardial feebleness if it becomes manifest upon exertion and is out of proportion to the degree of effort. Intermittence of the pulse may come and go in an elderly or aged individual without being an index to the state of the heart muscle, but habitual arrhythmia must be held to signify degeneration, especially of the auricles.

Increase in the area of deep-seated cardiac dulness is a highly important sign in the class of cases now considered; often the area can be shown to possess a quadrangular outline indicative of general hypertrophy with dilatation. It should be remembered, however, that marked increase is not always demonstrable, and hence careful measurements should be made in doubtful cases.

Auscultation may or may not yield valuable data, but in all cases of extensive degeneration I believe more or less evidence is to be obtained by close study of the heart tones. Weakness of the sounds in general is not so trustworthy, as a rule, as is a modification of individual tones. Thus the first tone at the apex may be short and valvular or muffled or accompanied by a short whiff of variable intensity (the murmur of muscular mitral insufficiency). The aortic second tone is very likely to be sharply ringing or clanging, and may be impure; it may be enfeebled when there is dilatation as well as hypertrophy of the left ventricle, and often the first aortic tone is accompanied or replaced by a bruit. Many times the pulmonic second tone is accentuated, especially when the left ventricle has begun to dilate and there is muscular mitral insufficiency.

In summing up it may be said that the diagnosis of chronic myocarditis is warranted if symptoms of cardiac incompetence develop in a person past middle age whose arteries and heart present clinical evidence of sclerosis, on the one hand, and of dilatation or hypertrophy with dilatation, on the other. If angina pectoris or that form of spasmodic, nocturnal dyspnœa, termed cardiac asthma, is present there can be no reasonable ground for doubt. But if chronic nephritis is associated and has led to high blood pressure or perchance dominates the scene, there may be much difficulty in deciding in how far the hypertension or the state of the heart muscle is responsible for the symptoms of cardiac inadequacy. There are, moreover, border-line cases, usually seen in persons nearing middle age, in which a positive diagnosis of myocardial degeneration is so difficult that it is better to be content with the diagnosis of cardiac incompetence, leaving the question of degeneration *sub judice.*

Group II. In this class are included those cases in which sudden death surprises an individual, who, although he has attained or passed middle life, yet has not dreamed there was anything wrong with his heart. It also comprises those elderly men who apply for life insurance, and not presenting symptoms of cardiac inadequacy, yet by reason of their years are naturally looked upon with suspicion.

In this group there are no distinctive symptoms on the side of the heart, else there would be no doubt of their real nature, or if perchance the breath comes a little less freely during exertion than it once did, this is attributed to the individual's portliness or sedentary habits. In some instances there are slight precordial pain and a sense of fulness which, being especially noticeable after eating, are attributed to indigestion. In most of these cases symptoms either are wanting or are so slight that they do not intrude themselves upon the subjective consciousness of the individual, who may be engrossed in large professional or mercantile affairs. The main fact that excites suspicion is his age, which presupposes the conclusion that his organs and tissues are no longer healthy. How, then, may one arrive at a diagnosis as regards his myocardium?

Either one must come to an opinion through exclusion of all other sources of the symptoms, provided such exist, or the physician must infer that because the applicant is fifty-five or sixty years old his myocardium cannot be any longer sound, and hence makes a diagnosis of chronic myocarditis on general principles, as it were. Not only is this unscientific, but it is unwise, since it may be possible for the subsequent long life of the individual to cast discredit on the correctness of the examiner's judgment.

Cases of this group test the skill and judgment of the physician and oftentimes leave him sorely perplexed. Very much depends upon the extent of deep-seated cardiac dulness which, therefore, must be determined with painstaking accuracy. It is not sufficient to conclude that because relative dulness reaches to and not beyond the left nipple it is normal. The transverse area must be measured, for if dulness is found to reach more than 9 or 10 cm. to the left of the median line at the level of the fourth rib it is increased, even though it does not pass outside the mammillary line.

If the individual is large-chested and percussion of the heart is correspondingly difficult and uncertain, much aid may be obtained by study of the aortic second sound. If this is especially loud and ringing, it may be held to denote either abnormal peripheral resistance or sclerosis of the aorta. In the former instance in particular, arterial tension is likely to be high, and hence hypertension of over 150 mm. Hg., taken in connection with an intense and ringing aortic second tone renders the conclusion of left ventricular hypertrophy almost inevitable and corroborates the conclusion based upon percussion; if hypertrophy is discovered after middle age, it warrants the inference of degeneration.

It is always well to auscultate these doubtful hearts immediately after some unusual exercise—as hopping, bending, stair-climbing, etc.—which has set the heart to beating violently. Not infrequently one will detect, under such circumstances, a faint, soft, yet distinct systolic blow at the apex which accompanies, but does not replace, the first tone. This is the murmur of muscular mitral insufficiency and indicates that the papillaries or some other portions of the muscular apparatus do not functionate perfectly, in consequence either of degeneration or of some degree of dilatation of the ventricle. Whatever its precise mode of production this murmur denotes that the ventricular wall is not equal to the strain to which it has been subjected.

If the individual is past forty years of age and displays marked arterial hypertension, especially if such increased blood pressure is associated with an impressively corpulent abdomen, the inference is justifiable that the myocardium is being overtaxed. Should the examination of the heart result at the same time in the detection of an increase in the area of deep-seated cardiac dulness, this finding should be taken to indicate not a large heart to fit a large body, but an actual hypertrophy; and when hypertrophy and hypertension co-exist there is degeneration of the wall in consequence of habitual overwork.

The difficulty of determining the question of whether or not chronic myocarditis exists is illustrated by the following case:

A man, aged fifty-three years, consulted me because of precordial pain, of an anginal character, which had come on at 9 P.M. a week before. He weighed 240 pounds, which, for his height of five feet eleven and a half inches, constituted veritable corpulence. His abdomen was particularly prominent, although panniculus adiposus was everywhere pronounced. The anginal seizure had required medical treatment and had recurred at intervals nearly all night. Since that time he had been conscious of a dull, oppressive substernal pain upon walking, but in other respects he considered himself well. Upon inquiry it was learned that in the afternoon immediately preceding his angina he had taken the eighth of a course of violent exercises intended to reduce his weight and had felt "completely done up." Examination of the heart was not easy on account of the thickness of the chest-wall, yet, by careful percussion, it was possible to determine general increase in the area of deep-seated dulness, especially to the left. The heart sounds were feeble and in particular the aortic second tone was not ringing. At the apex the first sound was short and valvular and accompanied by a faint whiff which, in the dorsal decubitus, became distinctly blowing. Systolic blood pressure taken with a Janeway apparatus registered 138 mm. Hg., and the pulse rate was between 90 and 100.

Here was a corpulent man, of fifty-three years, who presented evidence of cardiac dilatation that had resulted from strain and who gave a history of angina pectoris. The queries naturally arose, Was heart strain the only condition, or was there an underlying degeneration? did the attack of angina lend support to the latter inference, or was it due to the dilatation and not to coronary sclerosis? The answer to these questions was by no means easy and had to be found by inference and not positive data.

There was no evidence of arterial thickening and the aortic second tone was not ringing and accentuated. But blood pressure was

138 mm. Hg., the abdominal corpulence was marked, and, moreover, the heart walls had yielded to a degree of physical exertion which they ought to have endured without lasting ill effects had the myocardium been perfectly sound. Furthermore, the initial symptom had been angina and not dyspnœa. On the whole, therefore, it seemed reasonable to assume more or less degeneration as a result either of obesity or of chronic strain incident to sclerosis of the intra-abdominal vessels and increased blood pressure. Pulse tension was not very high, to be sure, but it was believed that the reading was lower than it would have been prior to the occurrence of dilatation and consequent feebleness of the left ventricle. Nevertheless, since this conclusion was open to doubt, it seemed the part of wisdom to diagnosticate only the dilatation and myocardial incompetence, leaving the question of degeneration an open one which would probably find answer in the results of treatment.

TREATMENT falls under two heads, in accordance with the two groups under which the subject has been considered:

Group I. In the cases presenting signs of inadequacy the management should be essentially the same as in waning heart power from any cause. In the second group of cases, on the contrary, treatment should be conservative, that is, designed to prevent the development of incompetence, either through acute strain or through gradual increase of the hypertension already existing and probably responsible for the hypertrophy present.

It would be manifestly impossible within the limits of this paper to enter into a detailed discussion of all the weapons at our disposal in our attempts to combat the foe that has attacked the citadel of life, and hence we should be content to consider only certain of these. We cannot hope to restore the structural integrity of the heart muscle, but instead we should endeavor to improve its potential capacity. To this end the first and indispensable requirement is rest. We cannot expect an overworked organ to regain its lost strength if it is permitted to go on overworking. The degree of rest necessary and the length of time during which this measure is to be enforced should depend largely upon the degree of inadequacy. It is my belief, however, that with the first appearance of myocardial incompetence the individual should be told emphatically that he must give his heart a rest.

When symptoms are mild it may suffice for the patient to remain in his room or on one floor, avoiding stairs and lying down a considerable part of the time; but when it is evident that the heart cannot bear even slight exertion the individual should be made to remain at absolute rest in bed. In some instances it may be necessary even to insist upon freedom from all mental effort of a fatiguing kind and from the excitement of conversation with family and friends. At the best, heart rest can never be complete, and yet as much relief from work as possible must be had in these cases.

As with the rigor of its enforcement, so with the length of time during which this mental and physical rest must be carried out, no fixed rule can be given. This should be determined by the exigencies of each case. But of one thing I feel convinced, namely, that absolute rest in bed for a long time is not as beneficial to a degenerated heart as it is to one which is the seat of valvular disease. For example, in the failing compensation of mitral stenosis it is absolutely necessary to restrict the flow of blood to the right heart by abolishing muscular movements and quieting the respiration. Hence, the patient is kept at complete repose in bed. Conditions are different with an inadequate myocardium, for if the heart is deprived of the aid to venous return residing in muscular contraction and deepened inspiration caused by exercise, it is compelled to carry on the circulation very largely through its propulsive force. For a while, at least, this is offset by the lessened number of its contractions and its greater total period of diastolic repose. But if this rigid rest in the dorsal decubitus be enforced for too long a time the benefit of lengthened diastoles is counteracted, as it seems to me, by several factors that are likely to be forgotten.

In addition to the pumping action exerted upon the contents of the systemic veins by the contractions of the voluntary muscles and diaphragm, which action greatly facilitates intra-abdominal circulation, exercise produces a dilatation of the intermuscular arterioles which causes a derivation of blood to the extremities and thus lightens the heart's work. Muscular exercise also increases the interchange of gases, and hence the supply of oxygen to the tissues, including that of the myocardium. If now muscular inactivity be too prolonged and too rigid in the class of cases now considered, there comes a time when the heart no longer profits by the rest in bed, as may be shown by a tendency to return of the symptoms which had disappeared soon after treatment by rest had been instituted.

Consequently if the heart muscle has not regained sufficient strength to admit of voluntary exercise it must receive the assistance which comes from massage and properly conducted resistance exercises. The former is often of considerable benefit, but should not be applied vigorously to the abdomen on account of the elevation of blood pressure caused by deep manipulation of the abdominal parietes. This portion of the body may be massaged gently, however, and then with benefit. It is often advantageous to have rubbing supplemented by passive rotation and flexion of the joints by the masseur.

It is in this class of cases that resistance exercises find their chief sphere of usefulness. Their modus operandi is not clearly understood, but to my mind their benefit comes from the fact of their supplying the assistance so imperatively needed by the degenerated myocardium, yet without accelerating its contractions as does active voluntary exercise. Certain it is that the degree of benefit to be attained may be measured by the effect upon the heart rate. They prove of valuable aid in the carrying out of the principle of rest in the management of these cases.

In this connection may be mentioned the so-called Nauheim baths. Probably no class of cardiopaths obtain as much benefit from this balneological treatment as do persons suffering from myocardial incompetence apart from valvular disease. Although not at all wishing to be thought an advocate of these baths at home by means of artificial waters in preference to those at Bad-Nauheim, still I believe, from thirteen years of personal experience with the employment of artificial waters, that very striking improvement will follow in many cases, provided the baths are ordered with skill and judgment. I do not approve, however, of the efforts being made by a certain commercial house to introduce their effervescing bath tablets into indiscriminate use, since I am persuaded that their unskilful use may be productive of positive harm.

The rules governing the administration of these saline baths cannot be discussed here, but I wish to emphasize the point that this mode of therapy should not be deferred until cardiac incompetence has become advanced. The sooner it is begun after the appearance of symptoms, the greater is the prospect of improvement. Furthermore, it is well, in most cases, for the patients to take a course of baths annually, since a degenerated heart which once has shown signs of inadequacy requires frequent and repeated bolstering.

Medicinal agents must receive but scant attention in this paper, since for the most part their administration is governed by the rules that apply to cases of decompensated valvular disease. The use of vasodilators can rarely be omitted, especially when the arteries are stiff, but I should like to emphasize the point that they should never be neglected when digitalis is being administered in a case showing pronounced hypertension together with myocardial incompetence.

Digitalis and its congeners may likewise be passed by with but very few words. They are probably indispensable at some time in this group of cases, but are to be handled with care and judgment and require careful watching.

Theoretically, strophanthus is the drug which should yield the happiest results in these cases, and I have often been much pleased with its effects. Nevertheless no one cardiac tonic is equally suitable to all cases, and it is actual trial that must serve as the guide.

Formerly, from theoretical considerations, I was strongly opposed to the employment of aconite in cardiac disease. Experience has convinced me, however, of its great utility in some cases of myocardial degeneration characterized by great hypertrophy together with dilatation. In such cases tincture of aconite controls the uncomfortable sense of palpitation which annoys some patients. When, on the contrary, the heart chambers are greatly dilated and inadequate, I believe this remedy is not beneficial, if indeed it be not positively harmful.

Cathartics form a class of medicinal agents which deserve special attention. Although capable of doing harm when pushed to the extreme for too long a time, I yet believe they are employed too gingerly by most practitioners. Whenever the portal system is much engorged, cathartics are indispensable. Stasis within the great intra-abdominal vessels acts as a dam to the circulation. Under such circumstances the heart is obliged to propel the blood stream against a barrier. To remove this hindrance we must either bleed or purge, and as frequent loss of blood by venesection would be disastrous we must administer cathartics.

These should be bland and unirritating, and a saline is ordinarily the best. Sulphate of magnesium answers very well in many cases, but my preference is for some aperient water containing sulphate of sodium as well as magnesium. The correct principle of its employment, in my opinion, is to administer the cathartic daily, because, in the first place, an individual with a degenerated myocardium should never be allowed to strain at stool, on account of his liability to dilate his ventricle, and, in the second place, since the main reason of the cathartic is to keep down portal stasis, its action should be continuous, so to speak, or, in other words, daily and not at considerable intervals. The tendency to stasis is ever present, and if attacks upon it are spasmodic and unsystematic, as it were, the vessels will remain engorged or become more so in spite of us.

As long as pulse tension is high, as in the cases of cardiac inadequacy associated with chronic nephritis, the cathartic affords relief, because it removes intestinal toxins as well as lessens resistance in the great intra-abdominal vessels and thus facilitates the work of the heart. When, on the contrary, blood pressure has become low and dilatation is predominant the daily purge brings relief, because of its diminishing the ever-growing engorgement of the abdominal viscera. It is indispensable, therefore, in all cases of degeneration with consequent insufficiency of the myocardium.

Finally, a few words in regard to the use of morphine in this class of cases. Few therapeutic agents are of as positive value as is this, when properly administered. Its chief merit consists in its ability to relieve the attacks of dyspnœa (cardiac asthma), which so often rob these cardiopaths of needed rest. Given in a small dose subcutaneously, $\frac{1}{10}$ to $\frac{1}{8}$ grain, late in the evening, morphine acts as an efficient heart stimulant and almost without exception will ward off the dyspnœa. In many persons it also exerts a satisfactory hypnotic influence and brings the much-needed repose and refreshment. Were this the only service rendered by this remedy it would make the agent well-nigh indispensable. But when administered in a small daily dose and in the manner recommended, morphine becomes a positive tonic to the inadequate heart and contributes powerfully to restoration of potential strength and prolongs life when recovery is impossible.

In the majority of cases a dose larger than $\frac{1}{8}$ grain is not necessary, and hence I regard it as a mistake to begin with $\frac{1}{4}$ grain, as is so often done. It is my invariable rule to follow the morphine next morning with a cathartic containing sodium, and when the bowels are kept open by this means I have rarely seen intolerance of the drug. I have known cases in which $\frac{1}{8}$ grain was administered hypodermically every evening for many weeks without the necessity of increasing the dose.

I have recently resorted to the hypodermic use of $\frac{1}{12}$ to $\frac{1}{6}$ grain of heroine for the relief of nocturnal dyspnœa, and have seen it followed by almost miraculous relief when such had not been obtained by morphine. I have never observed the same results from the use of morphine or opium by the mouth as by subcutaneous injection. The objection that dependence upon morphine may become a fixed habit is of no weight in a case of incurable heart disease, which, if restored to some degree of competence, yet cannot last for many years and oftentimes for not more than a few months.

Group II. The management of this class of patients contemplates not the restoration of cardiac sufficiency, since this has not been lost, but its preservation. In other words, treatment is now preventive, so far as such is possible. By the time the existence of myocardial degeneration is recognizable or at least presumable, it is probably too late to do aught toward its restriction, and, therefore, the aim of management must be to protect the weakened myocardium against injurious influences and to assist it in its struggle to maintain the circulation.

Our first duty, therefore, is to inform the individual of the exact state of affairs, lest if left in the belief that all is well with him he fail to co-operate by strict obedience to injunctions. Next, he must be warned of the danger attached to activities which involve heart strain and hence the liability to dilatation. Running to catch trains, the hasty ascent of long flights of stairs, arduous mountain climbing, the lifting and carrying of heavy weights, as hand-baggage, business activities involving much excitement or fatigue, etc., must be avoided. Absolute freedom from all injurious influences of this kind may not be practicable, and yet very much can be done in this direction by the person who is impressed with the harm that is likely to ensue.

The prevention of acute heart strain, important as it is, is in reality subordinate to prophylaxis as regards chronic overtaxing of cardiac power. This latter is brought about through the persistence or perchance increase of the hypertension so commonly seen in these cases. Prevention calls, therefore, for correction of all those factors of overfeeding, lack of exercise, and strenuous business life which make for high blood pressure and consequent augmentation of peripheral resistance.

In particular I wish to dwell on the value for these individuals of systematic and regulated medical gymnastics. These consist of breathing exercises, arm and leg movements, and contractions of the abdominal muscles, conducted in the presence, under the supervision, and by the assistance of a skilled medical gymnast, who keeps careful watch of the pulse and respiration, lest the exercises be so violent as to cause circulatory or respiratory embarrassment. The heart laboring against high blood pressure may be readily overstrained, but if, on the contrary, the exercises are at first light and easy the heart becomes accustomed to them and will at length bear vigorous physical efforts without undue acceleration of the pulse.

I doubt the wisdom of allowing an individual to carry out such a system of exercises alone, unless indeed the physician be certain of the potential integrity of the heart muscle and of the person's judgment. After the patient has learned the proper method of exercise and the amount of physical effort which his heart will endure, he may continue them without further assistance and direction by the gymnast.

Along the same line are golf, horseback riding, walking, the so-called terrain-kur of Oertel, light dumb-bell exercises, the sawing and splitting of wood, etc. The younger the individual and the greater the likelihood of a still elastic aorta and competent myocardium the more vigorous may be the daily exercise. But in all instances the physician should maintain supervision of the individual until convinced of the benefit and not harm resulting from the physical exercise. If the person displays the initial symptoms of myocardial inadequacy he should be permitted nothing more severe than the resistance exercises already advised for the cases in the first group.

Medicinal treatment has but small place in the management of these cases. The keynote of the situation is change of habits and the institution of a hygienic mode of life. Nevertheless a certain amount of drug therapy may be of service. If pulse tension is high and abdominal corpulence bespeaks high intra-abdominal blood pressure, cathartics are of great value. In such cases the work of the heart is materially assisted by an occasional brisk purge by calomel and a saline or the compound infusion of senna. In all patients displaying habitual constipation this should be corrected, as straining at stool greatly augments the harm resulting from the constipation. An occasional course of the laxative waters of some spa, as Carlsbad, West Baden, French Lick, or elsewhere, may prove very beneficial.

The administration of vasodilators is indicated only in those cases which display such an excess of arterial tension that it is but a question of short time before the heart muscle must yield to the strain. When such a dire event is imminent the various measures just advocated must be carried out promptly and with judgment, since delay and indiscretion may be tantamount to the proverbial locking of the barn-doors after the horses have been stolen.

A PROPER SHOE AS AN AID TO TREATMENT IN FLAT (OR WEAKENED) FEET.

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There is considerable confusion and misunderstanding in the minds of many of the profession in regard to flat-foot. The term as applied in many instances is misleading. The weakened foot goes through a series of changes, beginning with a disability known as pronated foot and ending with the deformity of rigid flat-foot (Figs. 1 to 7). The term flat-foot cannot be used to designate all the stages of this trouble, but should be restricted to one phase of the progressive weakened condition.

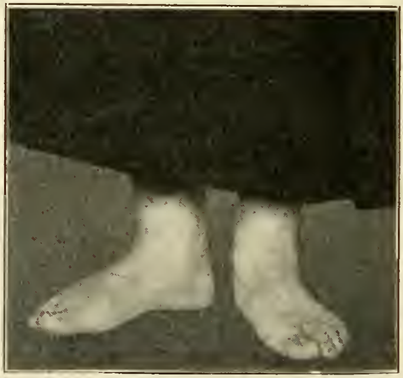

Fig. 1.—Weak and painful foot; slight pronation; arch not obliterated by weight-bearing.

Recently[1] I discussed the series as regards etiology, symptoms, diagnosis, and treatment, and stated that two of the chief aids to treatment are a proper brace and a proper shoe. Subsequently[2] I discussed fully the indications for a brace and described a form of brace which had been found useful in these weakened conditions. In the present article I wish to point out some of the advantages of a proper shoe, and describe its form and manufacture.

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[1] Albany Med. Annals, April, 1906.

[2] Ibid., September, 1906.

A brace is absolutely necessary at times and almost always beneficial; but, in order that a brace may have its full benefit, it should be used in conjunction with a proper shoe. The possibility of recovery and ultimately giving up of the brace is due, in large measure, to the use of a proper shoe; but I must emphasize the fact that, as all of treatment is not in a brace, neither is it in a brace even when used in conjunction with a proper shoe. Patients walk with their feet and not with a brace or shoe, and, therefore, they must strengthen their feet. Strength comes from a brace or shoe only as it is properly used. The physician can supply braces and shoes, but the patients must cure themselves. The physician can put the foot in a proper position, but whether the foot retains that position or not depends on strength of the foot, and strength of the foot can be brought about only by active exercise, gymnastics, etc., on the part of the patient.

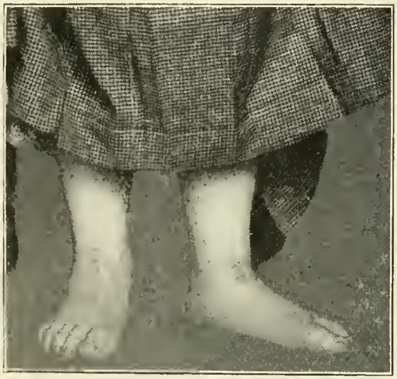

FIG. 2.—Weak and painful foot; pronation; obliteration of the arch by weight-bearing.

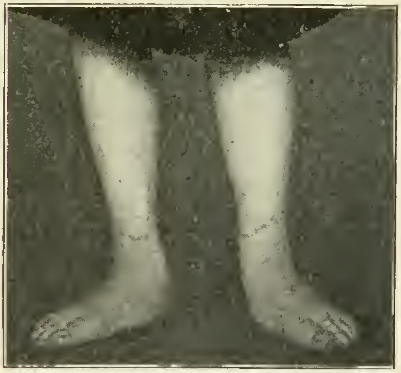

FIG. 3.—Markedly weak foot; painful; marked pronation; obliteration of the arch by weight-bearing.

In a former article the foot was compared to a machine, and it was stated that inasmuch as one of the causes of the breaking down of a machine is defective support, so when there is defective support to a foot in the form of an improper shoe, there is likely to be a breaking down of the foot. Furthermore, since when a machine is broken down it would not be a wise policy to repair the machine and leave a poor support, likewise there is little use in correcting a weakened or deformed foot and leaving uncorrected a poor support in the form of an improper shoe. It follows, therefore, that a proper shoe is indicated in all weakened conditions of the foot; moreover, a proper shoe is not only a therapeutic agent but also a prophylactic measure.

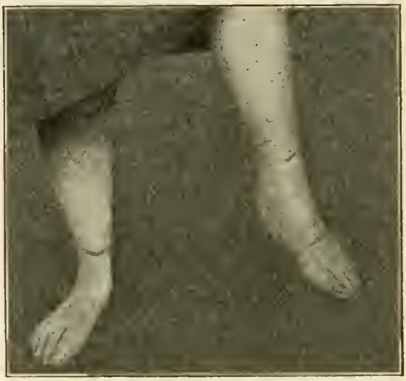

FIG. 4.—Markedly weak foot (same as Fig. 3); normal appearance of the left foot when the weight is removed.

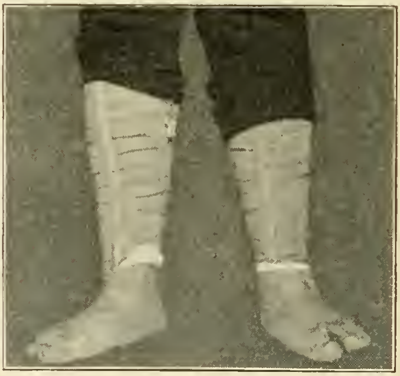

FIG. 5.—Flat and painful foot; not rigid.

The question might be asked, Why do we wear shoes at all? The human race, that is, the more civilized portion, has worn some form of foot covering for centuries, and, in many ways, the history of shoes is closely associated with the history of the race. Hill in his *History of English Dress*, in discussing the origin of clothes, says that it involves many principles of psychological interest. In the primitive state the factors which first prompted man to devise raiment and which tend to modify it are: protection (as becomes a more roving animal), climate, occupation, habits, and ornamentation. In a more advanced stage of civilization modifications arise through idiosyncrasies of taste, wealth, rank, and intercourse with other nations.

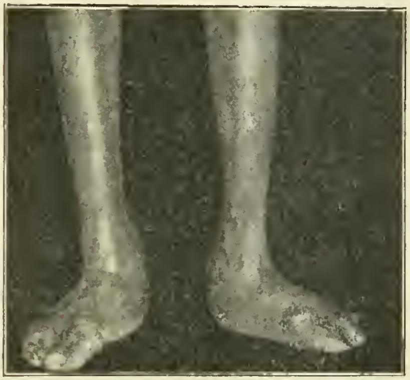

FIG. 6.—Rigid flat-foot; not painful.

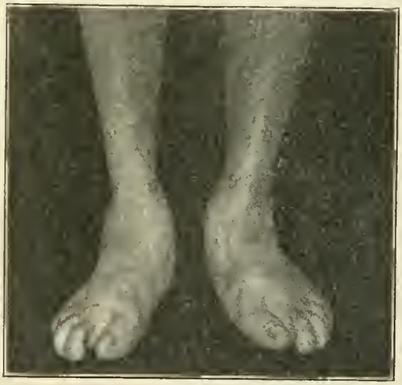

FIG. 7.—Rigid and painful flat-foot. Note the enlargement of the bones.

From the first, man was a roving animal and for protection to the feet devised sandals or, in colder climates, some form of moccasin. From these two elements, sole without upper and upper without sole, was evolved the modern shoe. In present times shoes have become not only a bodily necessity, but a necessity for fashion. In many instances their real use has been lost sight of and they are used principally to make the foot look well, or rather to conform to fashion's decree of what looks well.

The history of shoes shows a constant change from broad toe to pointed toe, from low heel to high heel. Sad to say, in spite of all the agitation that has been made at various times in favor of more perfect footwear, fashion still rules, and shoes are not much better adapted for the foot than they were hundreds of years ago. One might be tempted to cry out in a perversion of Madame Roland's famous passage, "Oh fashion, fashion, how many crimes are committed in thy name!"

It seems remarkable what a hold on the race the attempt to improve on nature in regard to the shape of the foot has taken. Read a description of the heroine's shod foot in any ancient or modern novel. Instead of describing a broad-toed, well-adducted boot, there is described the small, high-heeled, pointed-toed shoe. It requires only a little knowledge to read between the lines of the aching, burning feet inside and the nervous drain consequent upon their use.

In the time of the Plantagenets in England the toes of shoes became so long and pointed that laws were passed prohibiting their make; and the clergy were empowered to put under a curse those who extended their toes beyond the ordinary length. It was, however, the introduction of the fashion of broad-toed shoes which really put an end to the "Polish Points," rather than the majesty of the law. These new shoes became as exaggerated and absurd as the long, pointed shoes. Gladstone said of fashions: "It is a wheel going round and round, always puzzling you like a fireworks wheel, but always landing in a total negation of progress and with a strong tendency to the substitution of mere caprice for the real pursuit of beauty."

The rise and fall of heels might be used as an index of the times; there have been alternating low and high heels as fashion dictated. Sir Thomas Parkins, writing in 1714 in his *Treatise on Wrestling*, says: "For shame, let us leave off aiming at the outdoing our Maker in our true symmetry and proportion; let us likewise, for our own ease, secure treading and upright walking as He designed we should, and shorten our heels."

It is of interest to note that women have always worn shoes more inadequate for "walking abroad" than men. It is only as late as 1828 that boots for women's wear came into fashion.

In the early history of the United States, the travelling shoemaker journeyed from home to home making the shoes for the family. A last for the largest foot in the family, usually the father's, was whittled out first and a shoe made over it, and the last was then whittled down for each smaller foot in the family. Today the American shoes lead the world in excellence and cheapness of manufacture, but it is doubtful if they have improved much in their influence on the foot. In a comparatively recent article, Bradford calls attention to the fact that deformities of the foot change with fashion, thus giving rise to different deformities for different periods of history. The foot is capable of a great deal of distortion by shoes, but limitations in its usefulness must of necessity result. When the patient adapts himself to these limitations there is no trouble, but so soon as active use is demanded of the foot it sooner or later shows the effect of the strain. The common expression of breaking in a shoe really means breaking in the foot.

The reformer in shoes meets with some hard experiences. Custom and fashion have such a strong hold on the public that it is not an easy matter to persuade people that their shoes are at fault. In fact, some patients, especially women, cannot at once change from their old shoes to a proper shoe because the change is too great. The shoe habit is almost as bad as the morphine or whisky habit; when patients first give up the old and try the new way, they feel worse than they did under the old.

Another difficulty is found in patients—here again usually women—who try to wear two kinds of shoes: a proper shoe part of the time, and the old, narrow-toed, high-heeled shoe the rest of the time. One cannot serve the two masters of right and fashion when applied to modern footwear. It is difficult to understand why patients will choose to suffer from and treat calluses, corns, and painful feet instead of correcting that which is causing the trouble.

There is a class of patients who are afflicted with markedly weakened feet; oftentimes the arch has begun to fall, and they are wearing a soft, flabby shoe, resembling a moccasin with none of its advantages. The physician advises a stiff shoe with some support and one in which a brace could be used with benefit. The patients complain that they cannot get a shoe to fit and that they *cannot possibly* wear a stiff shoe: they have tried to do so repeatedly, but cannot. Of course, they cannot with any shoe they would buy themselves; but fit their feet with proper shoes and braces and the chances are that not only would they be able to wear the shoes, but the trouble would clear up at once.

How often the physician hears the complaint, "I cannot get a pair of shoes to fit me." Even when the patient gets custom-made shoes, he may not get comfortable shoes, for, as a rule, the custom-made shoes are constructed on the same principle as the ready-made shoes, only not so well made unless the patient is fortunate in his shoemaker. A prominent German writer, Hosebroek,[3] advises patients to get their shoes at a large establishment because they generally fit the foot better. The trouble is by no means always due to the shoe being too tight. In some ways it may be better to have a shoe too tight rather than too loose. Josh Billings is credited with saying: "There is one thing that can be said in favor of tight boots, they make us forget all our other troubles."

The chief faults with modern shoes are that they follow fashion in an attempt to improve on Nature in regard to the shape and appearance of the human foot; they cramp the foot; and they hold the foot in faulty attitudes. Bradford[4] calls attention to arboreal traits in the human foot manifest in infants, and states that these rapidly disappear with the use of shoes. Hoffman[5] draws some interesting conclusions from a comparative study of the feet of barefooted and shoe-wearing peoples. He concludes that the "forms, function, and range of voluntary and passive motion are the same in both up to the time of shoe wearing, after which progressive characteristic deformation and inhibition of function ensue. Here as in other instances acquired characteristics are not transmitted. The shoe wearers inherit the same type of foot as do those of barefooted races, and this type is changed only in so far as footwear modifies it." He experimented with savages who had never worn shoes and found that foot deformities begin to occur after only a few weeks of shoe wearing.

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[3] Zeit. f. Ortho. Surg., xi, 2.

The normal positions of the foot, both in the passive attitude and during activity, are well discussed by Whitman.[6] Bradford admirably describes the normal walk, and in an article entitled "Proper Footwear and the Treatment of Weakened and Flat Feet by Mechanical Devices for Maintaining the Adducted Position" Sampson[7] emphasizes the value of the adducted position. There are, therefore, certain postures which are normal whether the foot is at rest or in use, and if these normal postures are interfered with in any way the foot is placed in a false position and is consequently under a strain.

What, then, are the requirements of a proper shoe? In the first place it should be understood that there is nothing elaborate about a proper shoe; it is simply a shoe that supports the foot in its normal position when at rest and allows the normal motions to take place during activity. In order that these may occur the shoe must, of necessity, be an individual shoe made for that foot alone. As has been previously stated, the expression of "breaking in the shoe" really means breaking in the foot. I do not understand why people should use ready-made shoes any more than ready-made teeth.

A proper shoe should have a low heel with little or no spring to the sole, thus allowing the toes to come to the ground. They should be broad enough across the ball of the foot to give full play to the weight-bearing portion. The shank should be to the outer side of the shoe (where the weight of the foot comes) instead of in the middle, as in the ordinary shoe. The front part of the shoe should have the same angle of adduction as is normal to that individual foot. In the proper shoe there is no attempt made to improve on Nature, but when nature has been violated an attempt may be made at correction.

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[4] Boston Med. and Surg. Jour., November 24, 1904; Jour. of Med. Research, May, 1902; Amer. Jour. Orthopedic Surg., April, 1905.

[5] Ibid., October, 1905.

[6] Text book of Orthopedic Surgery.

[7] Johns Hopkins Hosp. Bull., 1902, xiii, No. 130.

Care should be taken, especially in weakened feet, to keep the heels of the shoes built up and not allow them to become worn off on one side. This is a very important point. I have recently seen a case illustrating this point. The patient was wearing a proper shoe and I could not understand why pain was present in the foot until I observed that the heels were worn off on one side. On correction of this fault the pain promptly disappeared.

To a certain extent the profession and the general public are becoming alive to the importance of proper footwear. Almost all the large shoe manufacturers are putting on the market shoes advertised as beneficial in flat-foot troubles. No attempt will be made to explain or even enumerate the individual features on which the manufacturers base their claims of a proper shoe.

Many of the shoes are excellent, but the great fault is that they are not shoes for the individual foot, that is, they are not custom-made. There are various shoes on the market which have a brace incorporated into the shoe. This is a bad idea, because when the brace is made into the shoe, the shoe becomes a splint to the foot.

What is, theoretically, an excellent scheme for throwing the weight of the body on to the outer side of the foot in walking has been devised by a Boston shoemaker. The shoe has a soft, flexible shank, and a square of rubber is inserted into the forward and outer portion of the heel of the shoe. The idea is that in walking the back of the heel will strike the ground first, then as the weight falls on the forward portion of the heel the rubber portion on the outer side gives and the weight of the body is thrown to that side.

I wish to describe what I shall call the Sampson last and shoe. Dr. Sampson in a paper, the title of which I have already quoted, discussed these fully from a mechanical standpoint; in the present article they will be considered more as regards their practical application.

The special features of the last are as follows: The last is shown in Fig. 8, *A*. It is so constructed that the line of body weight projected on from the middle line of the leg and ankle falls over the centre of the last. As regards this point, compare Fig. 8, *B*, the last of a modern fashionable shoe. The effect of weight-bearing on the two types of lasts is shown in Fig. 9. The shank is placed to the outer side of the last and there is no external longitudinal arch unless imprints show the presence of an "island foot," in which case the last is given a slight external longitudinal arch. There is little or no spring to the front part of the last, that is, it comes in contact with the ground for its whole length. The under surface of the last is concaved to correspond to the transverse arch of the foot. The last is thickest over the inner side corresponding to the position of the big toe (the thickest portion of the front part of the foot) and diminishes in thickness to the outer side. There is a fulness in the last corresponding to the situation of the big-toe joint; the toe of the last is rounded instead of pointed, and it has the same angle of adduction as the foot for which it is made. The outer edge is nearly straight.

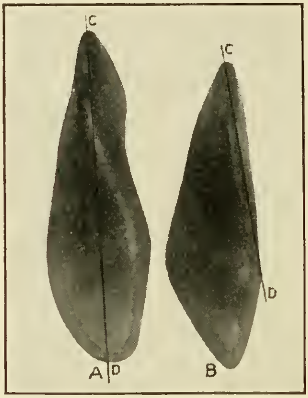

Fig. 8.—*A*, Sampson last. Note that the line of body weight, *C–D*, falls over the centre of the last. *B*, last of a modern fashionable shoe. Note that the line of body weight, *C–D*, falls to the inner side of the last.

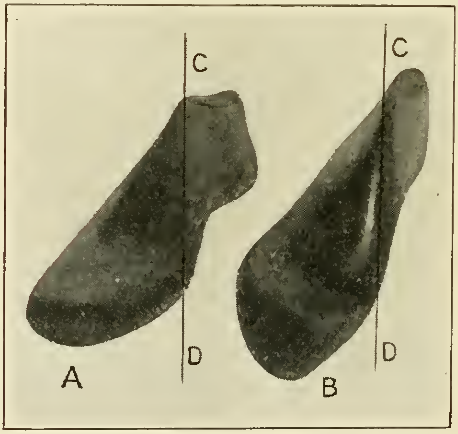

Fig. 9.—*A*, Sampson last; *B*, modern fashionable last. Note the effect of a force (body, weight) acting downward along the lines *C-D*. *A*, remains firm; while *B*, tilts to the inner side.

The shoe is made over this last and, of course, has the same shape and characteristics. The shoe is preferably a high, laced shoe. The heel should be low with a firm counter grasping the heel of the foot. There should be an extra-stiff counter in certain cases when a brace is used with the shoe, and there is a marked tendency to running over at the heel. As a rule, the shoe should have a stiff steel shank, and this is an essential when a brace is used; otherwise the brace would force the shank down and the support would be lost.

Nearly every writer on the treatment of flat-foot conditions mentions the value of a proper shoe, but only a certain number of these writers give the reader any hint as to what a proper shoe is and a still smaller number describe the manner of getting a proper shoe made. I wish to describe briefly the method which I employ in getting for the patient a proper last and shoe.

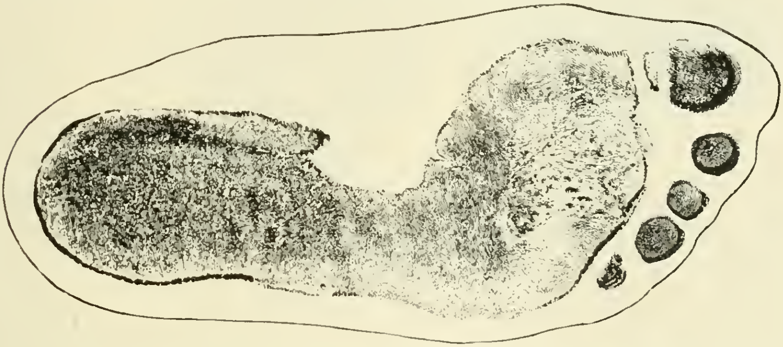

FIG. 10.—Impression of the weight-bearing portion of the foot, and a tracing of the outline of the foot.

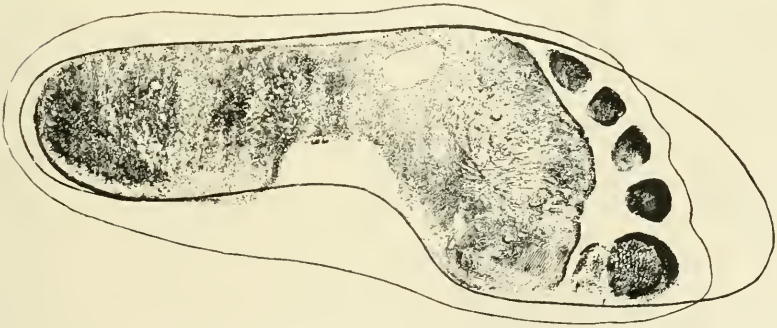

FIG. 11.—Inner sole pattern for the last, made with the imprint and the tracing as a guide.

The first thing to be obtained is an imprint of the foot. A mixture of 1 part of lampblack, 1 part of powdered acacia, and 3 parts of water (a mixture which can be readily washed from the foot) is thinly spread on a sheet of plate glass by means of an ink roller. The patient steps on this with one foot with his whole weight, thus blackening its under surface. He is then instructed to raise his foot, and after the glass has been replaced by a sheet of rough white paper to bring it down upon the paper and rest his full weight upon it. A tracing is then made around the foot. When the foot is placed upon the paper care should be taken that it is held parallel to the other foot, since in that position the feet have their normal amount of adduction. On the patient's removing his foot there is obtained what is shown in Fig. 10—an impression of the weight-bearing portion of the foot, together with a tracing of the foot. The imprint shows the full weight-bearing portion during activity and also the amount of adduction normally present. An inner sole pattern for the last is now marked out with the imprint and the tracing as a guide (Fig. 11). The custom shoemaker ordinarily blocks out the inner sole pattern from the outline of the foot alone, and consequently is apt to disregard both the weight-bearing portion of the foot and the normal adduction.

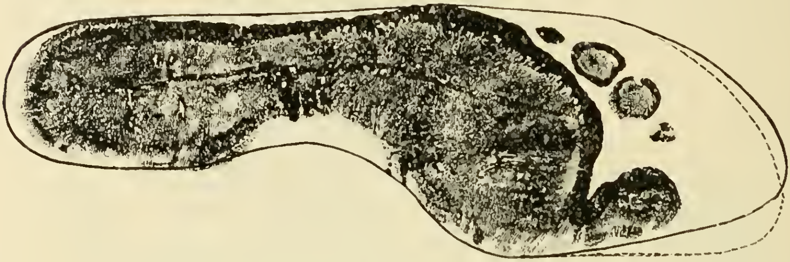

FIG. 12.—Illustrating the differences between the regulation shape of a shoe and a shoe with a straight inner edge.

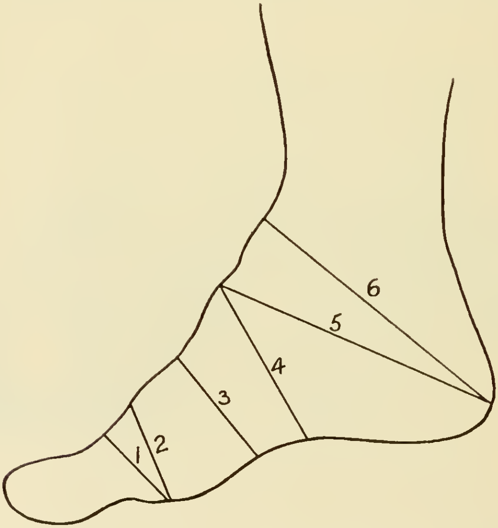

FIG. 13.—Illustrating the method of measuring the foot for a proper shoe.

With nearly everyone who has worn shoes there is a slight hallux valgus present. Theoretically, the inner side of the inner sole pattern should be straight to allow of adduction of the big toe, but ordinarily, unless there is marked hallux valgus calling for treatment (the wearing of a toe-post, etc.) it is customary to allow the toe of the shoe to conform more to the regulation shape, since it has been found that the toe will rarely move in to fill the vacant place that would be left were the edge made straight (Fig. 12).

The services of a shoemaker are now called in and measurements of the foot are taken as follows: (1) Circumference of the foot over the big and the little toes; (2) the ball of the foot; (3) the waist; (4) the instep; (5) the heel and the top of the instep, and (6) the heel (Fig. 13). Measurements are also taken for the ankle and the top of the shoe. These measurements and the inner sole patterns are now sent to the lastmaker.

The lastmaker has a model last in stock and he makes for the patient a last similar to the model last, but having an inner sole pattern and measurements corresponding to the patient's foot. The last is now returned to the shoemaker, who makes any little change that may be necessary to ensure a more perfectly fitting shoe, and the shoe is made. It will, of course, be understood that separate inner sole patterns, measurements, and lasts have to be made for both feet of the patient.

In conclusion, I would say that most excellent results have followed the use of a proper shoe such as has been described. There is almost always some difficulty on the part of the patient in becoming accustomed to the new shape; but in a few days the discomfort wears away and the freedom of motion and comfort consequent upon the use of properly constructed shoes soon cause him to become an earnest advocate of their use.

THE TREATMENT OF COMPRESSED-AIR (CAISSON) ILLNESS.

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While compressed-air illness, or caisson disease, is an affection which is seldom seen by the general practitioner, it is becoming of considerable interest on account of the growing necessity for increased transportation facilities and the consequent need for underground tunnels, bridges, and other structures which necessitate the sinking of caissons. The disease occurs as a result of work in compressed air at pressures greater than the normal fifteen pounds to the square inch, and is believed to be due to the presence of gas emboli in the circulation. A discussion of its causation, however, is beyond the scope of this paper.

Toward prophylaxis of the affection much can be accomplished by taking certain precautions. All applicants for work under pressure should be subjected to as rigid a physical examination as that considered necessary for an individual applying for life insurance. Preliminary to this examination the previous history of the candidate with regard to ophthalmic, nasal, aural, rheumatic, cardiac, pulmonary, hepatic, renal, and venereal conditions and cranial injuries should be ascertained. He should also be questioned as to whether or not he has ever worked in air under pressure, and, if the reply is in the affirmative, whether he has suffered from any of the symptoms of caisson disease. The subject should also be submitted to a thorough physical examination, and his urine should be tested for albumin and glucose.

The conclusions may be drawn that the laborer in compressed air should not be younger than twenty nor older than thirty-five years, that his muscular system should be, at least, normally developed, that his eyes and ears should be normal, and that his general physical condition should be in no way vitiated. It is usually the part of wisdom to disqualify, at least temporarily, subjects whose pulse is too rapid (above 100) or irregular, as well as those who give evidence of cardiac, arterial, pulmonary, hepatic, or renal disease. Alcoholism, unless the applicant exhibits the signs of this condition in its acute stage, is not a reason for disqualification, else it would be most difficult to obtain workers. Observation seems to show that the negro and the Anglo-Saxon races are less subject to the evil effects of air under pressure than are Italians, Poles, and other Eastern Europeans.

In considering the examination of candidates for employment a word should be said concerning status lymphaticus. It has been held by pathologists, who have made autopsies upon subjects dead of compressed-air illness, that status lymphaticus is often present, and consequently the signs of this condition should be sought as a part of the routine physical examination. Status lymphaticus is an obscure condition and one most difficult of diagnosis, but the following points are useful in its recognition: The lymphoid tissues should be examined for evidence of hypertophy. This examination should include a consideration of the superficial lymph glands (which often are but little enlarged) and an inspection of the throat for hypertrophied tonsils, lingual and pharyngeal tonsils, and adenoid growths. Splenic and thymic enlargements should be sought, and the heart and bloodvessels should be examined for hypoplasia, as evidenced by small size and thinness of the walls of the accessible arteries. According to Conner[1] the condition of the heart in status lymphaticus is variable; the organ may be hypoplastic or the left ventricle may be hypertrophied or dilated; in hypertrophy the pulse is small and of high tension. The body should be inspected for signs of infantilism, in which condition the beard and the axillary and pubic hair are scanty; the limbs, instead of being muscular and of masculine type, are rounded and resemble those of the female, and the figure, with regard to narrowness of the shoulders and breadth of the pelvis, may be more or less characteristically feminine. The height need not be below the normal; in fact, gigantism is sometimes present. Evidences of rickets and of thyroid enlargement should also be sought.

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[1] New York State Journal of Medicine, July, 1906.

After having passed a satisfactory examination the candidate should be subjected to a preliminary test, that is to say, he should be put under a pressure of from twenty-five to thirty pounds for an hour and a half; following this he should undergo a second physical examination, and should there be no reason for his disqualification, he may be allowed to undertake work in the compressed air, after having received certain warnings and advice, as follows: The air chamber should not be entered unless the subject feels perfectly well, and it is unwise to begin work on an empty stomach. Intoxicants should be used sparingly, or, better, not at all; but little water should be drunk while under pressure, and the laborer should obtain at least seven hours' sleep per day. As the pressure is raised the fulness in the ears, which is caused by the difference in the pressure outside the membrana tympani and that within this structure, may be relieved by swallowing or by closing the mouth, holding the nostrils, and performing the act of blowing the nose. This forces the Eustachian tubes open and thus equalizes the pressure. Emergence from the compressed air must be slow; at least fifteen minutes (and still better, thirty) should be taken to come out from a pressure of thirty pounds, the pressure being allowed to fall not faster than two pounds per minute. The laborers should come out of the pressure as follows: at the end of the shift they enter the air lock, the pressure in which is equal to that in the caisson where they have been working. The door between the caisson and the air lock should be closed and the exit valve of the latter opened sufficiently to allow a slow and gradual outflow of the compressed air. When this has escaped and the pressure within the lock has become equal to that outside the laborers should be liberated. After coming out hot coffee should be drunk freely, and warm, dry woollen clothing should be put on. All exposure to cold should be avoided and no undue exercise should be undertaken for at least an hour after emergence. Beer or other cold liquids should not be drunk for a considerable period after leaving pressure.

If the caisson is underground elevators should be provided to bring the laborers to the surface, for the climbing of ladders seems to predispose in marked degree to the occurrence of the evil effects of working in compressed air.

Individuals suffering from compressed-air illness usually exhibit the affection in one of four main types:

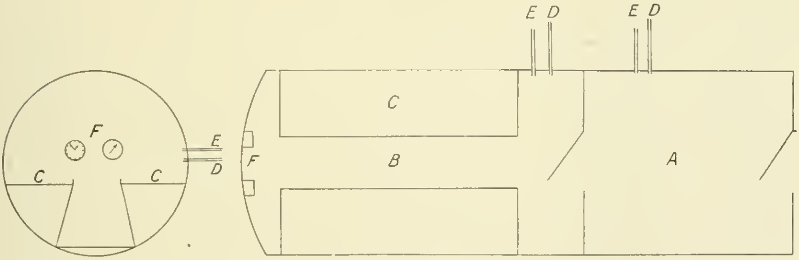
1. "*The Bends.*" This is the mildest form of caisson disease and is characterized by pains of varying severity. These are most common in the limbs; one limb or all may be affected, but the legs suffer more commonly than do the arms. The pains may be extremely severe; they are continuous, as a rule, but paroxysmal exacerbations may be observed. The pain is increased on motion, but there is no swelling, and tenderness is sometimes not notable. Itching of the skin is a frequent symptom. Abdominal cramps are not uncommon, and a condition termed "*the chokes*" is occasionally seen. This is characterized by cough, a sense of oppression of the chest, and some difficulty in respiration. The symptoms described, like all the manifestations of compressed-air illness, usually appear within an hour after emergence from the compressed air. Under treatment the pains disappear, as a rule, but they may be persistent, and sometimes reappear after apparent recovery.

2. "*The Staggers.*" This type of compressed-air illness is characterized by dizziness and staggering. At times the latter may be so marked that the patient falls, usually to one side or the other, unless he is supported. Vomiting may be present and the pulse may be rapid and weak. Spots may appear upon the skin of the abdomen, back, and limbs; these are purplish in color, and vary in size from that of a finger-nail to that of a quarter-dollar. There may be dimness of vision and nystagmus. The condition of the pupil is variable. The hearing is often diminished in acuity, but the patient will usually answer rationally upon loud questioning. There is seldom any mental disturbance. Pains in the limbs or abdomen may be present, and numbness of the extremities may be noted.

3. *The Paralytic Type.* The motor and other symptoms of this type of the affection are in no way characteristic. The loss of motility may be gradual or sudden, and there may be monoplegia, hemiplegia, or paraplegia; the last is, perhaps, most common. Sensation may be delayed or absent, or there may be irregular areas of anesthesia which sometimes occur without paralysis. The reflexes may be normal, lost, or exaggerated, and a spastic condition of the limbs has been noted. There frequently is retention of urine necessitating catheterization; more rarely there is paralysis of the bladder and the rectum, with incontinence. The mind, as a rule, remains clear. The paralyses may disappear permanently upon treatment; they may be temporarily relieved, to occur again, or they may resist all efforts. As a rule, however, they are not permanent, although months may elapse before entire recovery takes place.

4. *The Comatose Type.* In this type the most prominent symptom is partial or complete coma, which may appear suddenly after emergence from the pressure or may be preceded by other manifesta-

tions of the affection, more particularly those described under the second form of the disease. In the semiconscious cases active delirium may be present. The coma of caisson disease is in no way characteristic. The patient may suddenly become unconscious and die within an hour, or less, of cardiac or respiratory failure, the pulse growing weaker and weaker and the respiration increasingly slower until the end ensues. Comatose patients usually exhibit cyanosis of the lips and face. The skin is moist with a cold perspiration; the pupils may be normal, dilated, contracted, or unequal; their reaction may be sluggish and nystagmus may be observed. The pulse is often weak, thready, and irregular, and the respiration stertorous. There may be frothing at the mouth, nose-bleed, vomiting, sometimes with blood, and the rectum and bladder may be incontinent. The limbs may be flaccid or rigid, or there may be convulsive movements. The reflexes are in no way typical. The temperature may be normal or elevated. A fatal outcome is the rule in almost all instances in which absolute coma is present.



Plan and section of hospital lock. *A*, entrance chamber; *B*, hospital chamber; *C*, cots; *D*, compressed air supply; *E*, compressed air exhaust; *F*, clock, gauge, thermometer, and telephone.

In making the foregoing classification I by no means wish to draw definite lines of distinction between the different types, since the symptoms of the various groups are often so interwoven as to make arbitrary division quite impossible.

It seems probable that the indefiniteness of the symptoms of compressed-air illness is due to the fact that the gas emboli may lodge in any part of the nervous system and by their presence cause the clinical manifestations which become apparent, these latter being referable to the portion of the nervous system which is involved.

Treatment. In the treatment of all types of compressed-air illness the first consideration is recompression of the patient, that is to say, he must be subjected to the further influence of compressed air. For this purpose so-called hospital locks should be provided. Such a lock may consist of a horizontal cylinder made of one-half to three-quarter inch boiler iron; a very practical size is about twenty-five feet in length by seven feet in diameter (see figure). The lock should be divided into two chambers by a partition in which is an air-tight door opening outward. The open end of the lock should be fitted with a similar door. The double-chambered lock is the most practical type, for since both chambers are supplied with outlet and inlet valves, a means is thus afforded whereby the patient can be visited by an attendant without reducing the pressure to which the former is being subjected. Adjuncts which increase the efficiency of the lock are a couch, upon which the patient may lie; a telephone, by means of which he may communicate with the attendants outside; a clock, electric lights, and heating apparatus; the latter should be in the form of electric radiators, for steam radiators may possibly burst, a consequence which might produce most unpleasant effects.

Usually when the patient, if suffering merely from the first type of the affection, and especially if he has applied for treatment soon after the onset of his symptoms, is subjected to recompression he experiences very striking relief, the pains disappearing entirely long before the height of pressure in which he has been accustomed to work has been reached. The pressure is allowed to rise, even after relief is evident, to a height equal to that in which the patient has been working, and then he is "decompressed," that is, the pressure is slowly lowered to normal at the rate of about one-half pound per minute. Even slower decompression may be advisable in severe cases. Valves are obtainable which so control the outlet of air that the time necessary for the outflow of a given amount is the same whether the pressure within the lock be high or low. It must not be forgotten that it is necessary to supply the lock with a certain amount of fresh air after the inlet valve has been closed, otherwise the patient would be in danger of suffocation.

In treating instances of compressed-air disease it is unwise to subject the patient to the maximum pressure for more than five or ten minutes. When relief is experienced from the pain of an ordinary attack of the "bends" the patient is directed to move about the lock and to exercise and rub the affected part with a counterirritant ointment. In most of the mild cases the patient leaves the lock wholly relieved, but if there is a return of the pain recompression is advisable, the pressure being allowed to ascend until the pains disappear, when slow decompression is instituted as before. A third or even a fourth recompression may be employed if necessary, but usually the relief experienced from these subsequent treatments is temporary and slight in character. It is wise, if there is still continuation of the pain after the patient has been sufficiently recompressed, to advise thorough rubbing of the painful part with a counterirritant ointment or liniment. The following are suggested: capsicum oleoresin 2 parts, oil of turpentine 5 parts, oil of rosemary 5 parts, camphor 5 parts, olive oil 5 parts, petrolatum sufficient to make 100 parts; or menthol 10 parts, methyl salicylate 5 parts, hydrated chloral 10 parts, lanolin to 100 parts. The faradic current, applied as strong as can be comfortably borne, and the high-frequency current and vibratory massage may prove beneficial; hot baths, hot-water bags, and the electrotherm are very helpful adjuncts. Ironing the painful part with a hot flat-iron, a flannel cloth being interposed between it and the skin, is also suggested.

The persistent pains may be relieved by analgesics, such as salipyrine 10 grains every two hours, acetanilide 5 grains every four hours, or acetphenetidin 10 grains every three hours. These drugs should be given with care, and, if there is any tendency to heart weakness, they should be combined with citrated caffeine. Codeine may also be prescribed. Rarely are the symptoms severe enough to warrant the employment of morphine. In the management of all forms of the condition other than that characterized by pain, little in the way of specific treatment aside from decompression can be done.

Much, however, can be accomplished in the more serious types by the employment of symptomatic measures: when cardiac or respiratory weakness is evident hypodermic stimulation with strychnine, adrenalin chloride, caffeine, etc., should be resorted to. Artificial respiration may be indicated, and stimulation, by means of enemas of hot coffee, is always advisable in comatose cases. All these measures may be applied by attendants while the patient is under pressure in the hospital lock. Sometimes the results attained, even in patients in coma, are surprisingly good. Oxygen may be administered if there is respiratory failure, but should not be given while the patient is under pressure.

The after-treatment consists in careful nursing, feeding with nourishing food, and the use of tonics. Patients affected with persistent paralysis should receive the routine treatment of the chronic stage of peripheral neuritis. If the patient is confined to bed the prophylaxis of bed-sores is an important consideration, and if catheterization is necessary it is needless to state that this procedure should be conducted with the most thorough asepsis. The paralyzed muscles will be benefited and the patient's general condition will be improved by the systematic employment of galvanic and faradic electricity, massage, passive movements, and appropriate hydrotherapeutic measures.

A STUDY OF FIVE HUNDRED CASES OF PLEURISY

OCCURRING AT THE PENNSYLVANIA HOSPITAL.

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The patients upon whom this report is based were admitted to the Pennsylvania Hospital from the years 1896 to 1905, the five-hundredth case being completed with the end of October, 1905, thus covering almost exactly a decade. I am indebted to all the visiting chiefs, medical and surgical, who have allowed me the use of their material and encouraged the investigation.

In this series only inflammatory conditions of the pleura have been included, the transudates, such as at times occur in cardiac and renal disease, are not considered. So, also, cases of chronic thickened pleura without symptoms or physical signs (other than impaired percussion note or diminished breath sounds) were deemed outside the limits of this report. In the course of the investigation of the hospital records a large number of cases diagnosed as pleuropneumonia were encountered and analyzed, and those which appeared to be frank croupous pneumonia with but a small patch of pleurisy coincident (as evidenced by a friction rub heard only over the consolidated area) were discarded. All pneumonic cases in which the pleurisy yielded effusion, or which distinctly preceded or followed the pneumonic process, are included.

It is to be borne in mind that all statistical studies are notoriously variable, and even at times contradictory, as must needs be the case when one considers how different are the sources from which such statistics are derived. The hospital whence this series is collected is in the most crowded portion of a large city; it lies not far from the river front; the slums are almost at its doors; and by far the greater portion of the foreign and negro population of the city live in its immediate vicinity. The hospital receives scarcely any pay patients. The wards are filled with the poorest and most destitute class, native and foreign, whose resistance to disease from the many exigencies incident to poverty, or to evil living, is reduced to the lowest terms.

It is obvious, then, that a study of this series could only be compared with others made under similar conditions, and that it in no way represents what might be termed an average series of cases. Certainly not in private practice could one expect to find such numerous and remarkable complications, and the long-neglected and distressing conditions which here are often added to the difficulties of treatment.

Prevalence. The 500 cases occurred in a period of ten years, the number increasing yearly, in relation to the total number of patients admitted, in quite uniform proportion.

Year.	Medical cases.	Pleurisy.	Per cent.
1896	1,128	20	1.77
1897	1,272	25	1.96
1898	1,401	34	2.42
1899	2,001	38	1.90
1900	2,100	49	2.33
1901	2,118	66	3.11
1902	2,471	70	2.83
1903	2,564	64	2.50
1904	2,275	65	2.86
1905	2,066	74	3.53
	19,396	505	2.52

Norris' series of 500 cases of croupous pneumonia from the same hospital[1] occurred in four years, giving very fair means of comparison of the relative frequency of these diseases.

MENSAL FREQUENCY.

Winter: December, January, and February	138	
Spring: March, April, and May	156	
Summer: June, July, and August	120	
Autumn: September, October, and November	89	
First six months		304
Second six months		201
		505[2]

Pleurisy is, as may be seen from this table, more or less prevalent at all seasons of the year, but with decided tendency toward the colder months, and still more decided tendency toward the damper and wetter months. In the cold winter months, when snow and slush are frequent and winds high, the average is 52. In April, for some reason, there is a drop to 43, which is difficult to explain except by the element of chance which obtains in studies of a limited number of cases; but in May it rises to the highest total for the year, 62, to fall again in June to 43. After this time the average drops through the summer and early fall, reaching its lowest in October (20), and in the next two months rises again to 34, which is that of late summer, to take a larger rise at the beginning of the colder weather.

VITAL STATISTICS. In examining these tables we see that in children males and females are equally liable to the disease, but that after fifteen years of age, at which time their modes of life begin to differ, men are affected four times as frequently as women.

Regarding age, the table shows the remarkable fact that over one-third of the total number occurred in the decade between twenty and thirty years, and almost three-fifths in the score of years from twenty to forty. The ages are very variable, the youngest affected being but eleven months old, while at the other extreme of life two persons contracted pleurisy at the age of seventy-two.

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[1] AMER. JOUR. MED. SCI., June, 1901.

[2] The cases occurring in November and December, 1905, are added to complete the ten years.

Among the empyemas, of which it would seem of interest to make a separate table, we find that the tendency to pus formation appears to be much more marked in the young, and steadily decreases in later life. Leaving out the first five years, as there are no medical cases for contrast (the hospital not receiving medical cases under seven years of age), we see that in the next five years 50 per cent. were empyemas. In the next five years 43 per cent.; in the next five years 29 per cent.; in the next ten years 15 per cent.; in the next ten years 11 per cent., and in the next ten years 9 per cent. After fifty years the rate rises again to 17 per cent. in the next decade, and from sixty to seventy years 14 per cent.; but after seventy years there is no empyema in the 6 cases.

From fifty years on, however, the total number of cases is so relatively small that conclusions cannot be accurately determined. It may be of interest to note that in the ages from twenty to forty, through which period tuberculosis is most frequent, the empyema ratio is comparatively low.

Regarding empyema, men are affected three times as frequently as women, and here again the third decade of life furnishes the greatest number of cases, though not nearly in such marked degree as in the non-purulent cases.

Regarding the role which alcoholism is believed to play in the predisposition to disease, 102 of the 500 cases gave a distinct history of habitual indulgence in, or of excessive addiction to, alcohol. This is a high number when we consider the fact that from a number of the foreign patients a history could not be obtained, and that probably others deliberately concealed their excesses in this respect.

Males	389
Females	111
	500
Boys under fifteen years	25
Girls under fifteen years	23
Children	48
Adult men	364
Adult women	88

ALL CASES.

	No. of cases.	
Under five years of age	11	27
Over five and under ten years of age	16	
Over ten and under fifteen years of age	21	74
Over fifteen and under twenty years of age	53	
Over twenty and under thirty years of age	180	
Over thirty and under forty years of age	106	
Over forty and under fifty years of age	68	
Over fifty and under sixty years of age	32	
Over sixty and under seventy years of age	7	
Over seventy and under eighty years of age	6	
	500	

EMPYEMAS.

Males	70
Females	25
	95

	No. of cases.	
Under five years of age	11[3]	19
Over five and under ten years of age	8	
Over ten and under fifteen years of age	9	24
Over fifteen and under twenty years of age	15	
Over twenty and under thirty years of age	27	
Over thirty and under forty years of age	12	
Over forty and under fifty years of age	6	
Over fifty and under sixty years of age	6	
Over sixty and under seventy years of age	1	
	95	

NATIONALITY. Owing, as we have said, to the situation of the hospital and its class of patients, no general conclusions are warrantable from this table; but it must be surprising, nevertheless, to find that considerably less than half of our cases are native born, and that only one-third are white Americans—which may afford additional cause for reflection on the present increase of immigration.

The negro race furnishes more than one-eighth of the entire number of cases.

United States (white)	169
United States (colored)	65
Italy	152
Russia	37
Ireland	31
Austria	12
Germany	8
England	6
Greece	5
Rest of Europe	11
Canada	2
West Indies (colored)	2
Total	500

DURATION OF DISEASE PREVIOUS TO ADMISSION. Only one-seventh of the patients were admitted in the first three days of the disease; half came to the hospital between the fourth and the fifteenth days, with a slight preponderance to the latter half of the time. The poorer classes are indeed loath to enter a hospital, and they endure much before seeking such relief. In a later table it will be seen how serious is their delay. Twenty-two cases developed in patients already in the hospital (mostly empyemas following pneumonia).

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[3] Children under seven years of age are not admitted to the medical wards.

DURATION OF DISEASE ON ADMISSION TO THE HOSPITAL.

Less than twenty-four hours	8	
Over one day and less than four days	64	
Over four days and less than eight days.	122	
Over one week and less than fifteen days	127	
Over two weeks and less than twenty-two days	48	
Over three weeks and less than one month	44	
Over one month and less than three months	40	
Over three months and less than six months	6	
Over six months and less than twelve months	5	
One year or over	5	
		469

DEVELOPED IN THE HOSPITAL.

Empyemas (following pneumonia)	15	
Pleurisies (following typhoid)	5	
Other cases	2	
		22
Unknown duration	9	9
Total		500

Occupations. Here is well illustrated the fact that occupation predisposes to disease. Attention should be called to the relatively large number of drivers and stevedores, besides the common laborers, all of whom lead exposed lives, and a majority of whom are addicted to alcoholic indulgence, even if not actually intemperate. Among the indoor occupations we find not less than 24 tailors —a graphic illustration of the evils of the sweatshop. Cooks and waitermen (nearly all colored) are well represented, and though this hospital is far removed from the factory district we find 9 operators; while among the comparatively few printers we have 8 cases. A number of persons lead lives which cannot be classified under either head, and I think that among this number pleurisy seems relatively frequent.

OCCUPATIONS OUTDOORS.

Laborers	168
Stevedores	13
Drivers	15
Sailors	5
Other outdoor occupations[4]	20
	221

OCCUPATIONS INDOORS.

Housework	65
Tailors (sweatshops)	24
Waiters	12
Cooks	11
Printers	8
Factory hands	9
Plasterers and paperhangers	5
Other indoor occupations[5]	63
	197

[4] Includes farmers, police, peddlers, fishermen, etc.

[5] Includes barbers, clerks, salesmen, janitors, upholsterers, bartenders, etc.

OCCUPATIONS MIXED.

Children	46
Other mixed occupations[6]	36
	82

Past Medical History. One hundred and forty-eight gave a history of previous pulmonary trouble. This I think is a remarkably high percentage—almost a third, if we leave out the children under fifteen years of age. A rheumatic history was obtained in 46 cases, and 22 cases admitted specific disease; the latter proportion, it may be presumed, falling decidedly short of the actual number of syphilitics among the series. Of the acute infections only those occurring within the previous six months are admitted as having any bearing on the subject.

PULMONARY DISEASES.

Pneumonia (one attack)	53
Pneumonia (two attacks)	3
Pneumonia (three attacks)	2
Influenza (one attack)	18
Influenza (two attacks)	2
Old empyemas	5
Pleurisy (one previous attack)	40
Pleurisy (two previous attacks)	6
Pleurisy (three previous attacks)	1
Phthisis	9
Chronic bronchitis	4
Asthma	5
	148

GENERAL DISEASES.

Rheumatism (acute or subacute)	42
Rheumatism (acute, several attacks)	4
Syphilis (19 men and 3 women)	22
Plumbism	3

ACUTE INFECTIONS, RECENT, AND INFLAMMATIONS WITHIN SIX MONTHS.

Typhoid fever	8
Measles	2
Cholecystitis	1
Peritonitis	1
Perinephritic abscess	1
Hepatic abscess	1
Otitis media	1
Colitis	1
Tropical dysentery	1
Malaria	2
	19

Exciting Causes. The actual determining cause of the attack is here presented in the 233 cases in which such information could be elicited by either subjective or objective means. In connection with croupous pneumonia it is to be noted that 48 out of 52 cases of pleurisy following that disease were empyemas. In only 30 cases could direct evidence of tuberculosis, either pulmonary or miliary, be demonstrated antemortem. Traumatism accounts for 15 cases, and 12 cases developed immediately upon or after a debauch. The rather high number of 42 followed directly acute or subacute bronchitis, and perhaps some tuberculous cases may have been placed in this group, notwithstanding efforts at accuracy. Five cases occurred late in the course of typhoid fever, and a like number complicated acute articular rheumatism.

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[6] Includes carpenters, painters, plumbers, porters, hostlers, etc.

Exposure to cold, dampness, or sudden chilling	65
Following acute or subacute bronchitis	42
Following croupous pneumonia (of which forty-eight were empyemas)	52
Following traumatism (principally falls, broken ribs, and blows)	15
Occurred in the course of typhoid fever	5
Occurred in the course of acute rheumatism	5
Following phthisis (clinical, not with autopsy records)	14
Following a debauch (immediately)	12
Occurred in the course of double tertian malaria	1
Following influenza	2
Following acute tonsillitis	2
Following obstinate constipation	2
Following obstinate vomiting	2
Following appendicitis	2
Following cholecystitis	1
Following pericarditis	1
Following miliary tuberculosis	1
Following gastritis (acute)	1
Occurred in plumbism	1
Occurred in secondary lues	1
Following subdiaphragmatic abscess	1
Following hepatic abscess	2
Following abscess of chest-wall	1
Following carcinoma of breast	1
Following anesthesia	1
	233

Onset. A chill is noted in almost one-third of the cases. Multiple chills occurred in 3 cases. Pain is, of course, the predominant symptom, and is stated in 433 cases, in 3 of which the pain was entirely abdominal, and in 8 others occurred in both chest and abdomen. Cough is also a constant symptom and is mentioned in 369, while actual dyspnœa occurs 272 times. Vomiting is frequent (37) and epistaxis is not rare. In 3 children the attack began with convulsions. Of the side principally affected we note practically no difference—242 right as to 228 left, and 30 double pleurisies.

Chill	148
Multiple chills	3
Pain in chest	430
Cough	369
Dyspnœa	272
Diarrhœa	5
Vomiting (one persistent)	37
Epistaxis	16
Pain in abdomen (3 only in abdomen, not in chest)	11
Convulsions	3
Syncope	2
Delirium	2
Hemoptysis	1
Abdominal distention	1
Coma (alcoholic)	1
Great tenderness of chest	2

FRICTION SOUNDS were noted in 244 cases, in only 183 at the time of admission; but this fact is accounted for by the great number of patients who came to the hospital with large effusions of considerable duration. It appears that frictions during absorption are heard twice as frequently in left-sided effusion, and it is reasonable to think that the position and movement of the heart may play a part in its production.

Pleuropericardial rubs were noted 16 times, in 1 case the pleuritis being limited to the right side. Diaphragmatic friction are noted 6 times. One friction which had lasted two months was still present when the patient left the hospital, another lasted for five weeks, and in a third patient a friction rub existed for eleven weeks.

	Right.	Left.	Both right and left.	Total.
On admission	87	83	13	183
Developed during absorption or after aspiration	18	37	..	55
Pleuropericardial friction	1[7]	12	3	16
Diaphragmatic	4	2	..	6

EFFUSIONS. Three-fifths of the cases were serous effusions and one-fifth were purulent. In the other fifth no effusion occurred. In classifying the amount of effusion, the angle of the scapula was considered as one-third of the chest, the level of the scapular spine as two-thirds; all effusions above the spine were termed massive, while all below the angle were classified as basal. While not anatomically accurate, this classification appeared to be the simplest, and sufficiently correct for general comparison.

As might naturally be expected from the position of the heart, large effusions are more frequently left-sided.

On the right side there were 92 effusions of less than two-thirds and 105 two-thirds or more. On the left side there were 70 effusions of less than two-thirds and 110 two-thirds or more.

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[7] Undoubted.

Effusions.	Base.	1/3	2/3	Massive.	Total.
Right	38	54	69	36	197
Left	30	40	62	48	180
Right and left	7	12	2	3	24
	75	106	133	87	401

	Cases.	
Dry pleurisies	90	
Serous effusions	315	500
Empyemas	95	
Hydropneumothorax	4	
Pyopneumothorax	4	
Loculated empyemas	5	
Pulsating empyemas	2	
Pericardial effusions	4	

CARDIAC DISPLACEMENT. This sign was by no means as frequently mentioned as others, but notes were made in 216 cases. From the table will be seen the great variety of displacement possible. In those left-sided effusions in which no apex beat could be heard or felt in any part of the chest, and in which the sounds at the base of the heart were loud, it seems possible that there was actually posterior displacement of the apex to a limited degree; 17 such cases have been noted under that heading. When the lung has been compressed and no longer offers any support for the pericardium, it appears that merely by its own weight the heart may move away from the chest wall.

By far the greater number of cases in which marked displacement occurred were left-sided effusions. One left-sided case with two-thirds effusion had the apex in the normal position. In one massive effusion cardiac pulsation was seen, felt, and heard below and within the angle of the left scapula. One case had the apex beat in the fourth space beyond the right border of the sternum, and one case had the entire heart area to the right of the sternum, refused aspiration, and the heart gradually returned to its normal position in the fifth week.

	Up.	Up and to right.	Up and to left.	Down and to right.	Down and to left.	To right.	To left.	Post.
In right effusions	4	..	1	..	15	..	77	
In left effusions	9	20	..	6	..	56[8]	..	17
In right and left	3	2	1	..	..	..	5	

TEMPERATURE. In tabulating the temperature changes, it seemed advisable, for better comparison, to separate the cases into the acute, subacute, and more remote or chronic cases. Accordingly all cases admitted in the first week are classed as "recent," in the second and third weeks as "intermediate," and later than the third week as of "long duration." In 54 cases, 20 of which were empyemas, no temperature chart could be found. In the vast majority of cases the maximum temperature range was over 100° and less than 104°, and this held good for each subdivision as well. As might be expected, the greatest temperature reaction occurred in the most recent cases; 42 of the 60 cases exceeding 104°, being

[8] Nine beyond sternum.

in the first subdivision, whereas in the 60 cases whose maximum lay between normal and 100° only 24 belonged to this class. In 21 cases (only 4 of which were admitted in the first week) a subnormal temperature existed throughout the disease.

	Recent, 1st week.	Intermediate, 2 and 3 wks.	Long, over 3 wks.	Total.
Subnormal	4	5	12	21
Normal and below 100°	24	19	17	60
100° and below 102°	64	42	45	151
102° and less than 104°	81	38	35	154
Over 104° and less than 105°	23	10	6	39
Over 105° and less than 106°	15	1	1	17
Over 106°	4	0	0	4
	215	115	116	446

The highest temperature observed, 108.8°, occurred shortly before death in a maniacal patient who jumped from a window and was greatly shocked, though escaping without other injury. The lowest temperature observed, 94.6°, occurred in an alcoholic suffering from shock, fractured rib, and submersion, having fallen from a wharf into the river while intoxicated; he recovered. A distinctly hectic temperature was noted in 21 empyema cases, 11 tuberculous cases, and 2 simple serous effusions (1 in a child). A typical crisis was noted in 7 cases complicated by croupous pneumonia and in 1 with no pneumonia. Lysis was observed in 5 cases complicated by croupous pneumonia. Five cases of chronic empyema were absolutely afebrile. Five cases of long duration with high and irregular temperature on aspiration showed no pus. Two cases, undoubtedly tuberculous, were distinctly not hectic.

Aspiration. The amount aspirated usually was from 1000 c.c. to 2000 c.c., and but one aspiration was, as a rule, necessary. In only 20 cases the total amount of serum removed (by one or more aspirations) was over 3000 c.c. In all cases in which pus was found on aspiration, every effort was made to obtain consent for operation, and fortunately in nearly every instance was successful. The large number of exploratory aspirations made are not included in this table:

	1 asp.	2 asp.	3 asp.	4 asp.	6 asp.
Serum	124	25	7	4	0
Pus	33	4	1	..	0
Seropus	6	..	..	..	0
Serum, then pus	..	5	..	..	1

Hemorrhagic serum 8, bloody pus 3, aspiration followed by chills in 2 cases.

	Times.
Less than 500 c.c. removed	55
500 c.c. less 1000 c.c.	46
1000 c.c. less 2000 c.c.	94
2000 c.c. less 3000 c.c.	37
3000 c.c. less 4000 c.c.	8
4000 c.c. less 5000 c.c.	2

A total of over 3000 c.c. removed in 20 cases.

Largest: 9300 c.c. (6 asp.), 8700 c.c. (3 asp.), 7900 c.c. (1 asp.), 5900 c.c. (3 asp.).

Amounts: Largest amount at 1 asp. 4500 c.c.

Complications. The list is amazingly long and varied, over 50 different complications being noted, many of them sufficiently severe to add gravely to the prognosis. It seems scarcely credible that one-half of the cases were complicated, and tells graphically of the wretched class of patients which this hospital receives.

Of the more frequent complications, a word of explanation is necessary.

Nephritis heads the list, such a complication occurring 106 times. Only such cases as showed hyalogranular or granular casts are admitted, the presence of minute amounts of albumin, with or without hyaline casts and cylindroids, not necessarily indicating more than renal irritation. Under urine analysis this subject is more fully considered.

Croupous pneumonia occurred 56 times, either just antecedent to, during, or immediately following the pleuritis, and in every case contributed to the gravity of the condition, yet some remarkable results are recorded, not less than 3 cases having double pneumonia with empyema of one side, making perfect recoveries. One case had croupous pneumonia on one side and a large serous effusion on the other.

Bilateral pleuritis occurred 30 times, and I think may justly be considered as a complication, often most serious. Most frequently there was a large or massive effusion on one side, with a dry pleuritis or small effusion at the base of the opposite side, but in some instances there was quite a large effusion upon each side. In one case which came to autopsy an inflammatory effusion occupied one pleural cavity and a simple hydrothorax the other.

Phthisis or miliary tuberculosis could only be convincingly proved antemortem in 30 cases, though naturally suspected in many others. At autopsy it was frequently found, but almost always in those cases in which it had previously been diagnosticated, as they were the cases in which the death ratio was greatest.

Pericarditis occurred 9 times (in 2 cases of which there was only *right*-sided pleurisy). Acute endocarditis was found but 4 times. Pneumothorax is noted in 8 cases, 4 each with empyema and with serum. In 1 instance the air was admitted during aspiration. Abscess of the lung was found in 4 cases, all empyemas, and but 1 case of tumor occurred, probably a carcinoma. Abscess of the liver was found 4 times. Bronchopneumonia is comparatively infrequent, but 11 cases presented that complication. Other conditions will be found at length in the accompanying table:

COMPLICATIONS.

Total cases with one or more complications, 249.
Total complications noted, 360.

(*a*) Pulmonary.	Empyemas.	Serous	No effusion
Croupous pneumonia	23	25	8
Bronchopneumonia	1	6	4
Phthisis	8	16	3
Diffuse bronchitis	0	7	6
Abscess of lung	4		
Pneumothorax	4	4	
Miliary tuberculosis	0	3	
(*b*) Cardiac and Vascular.			
Acute endocarditis	2	1	1
Chronic endocarditis, v. h. d.	2	9	0
Pericarditis	4	5	0
Myocarditis	1	1	1
Arteriosclerosis	0	2	0
Aneurysm	0	1	0
Thrombosis (fem.)	0	2[9]	0
(*c*) Renal.			
Nephritis	20	63	23

(*d*) Infectious Diseases.—Typhoid fever 4, malaria 2, syphilis 1, measles 2, varicella 1, erysipelas 1, dysentery 1.

(*e*) General Diseases.—Acute rheumatism 6, exophthalmic goitre 1, sciatica 1, cirrhosis of liver 1, locomotor ataxia, 1.

(*f*) Intoxications.—Plumbism 2, iodism 1, septicemia 4.

(*g*) Special Inflammations.—Otitis media 5, parotitis 1, tonsillitis 6, pharyngitis 1, cholecystitis 5 (4 in right-sided pleurisy), perihepatitis 2 (in right-sided pleurisy), acute gastritis 1, peritonitis 1, splenitis 1, salpingitis 1, endometritis 2.

(*h*) Dermatological.—Urticaria 1, erythema general 1, purpura 2.

(*j*) Miscellaneous.—Obstinate constipation 1, prolonged diarrhœa 4, acute alcoholism 4, mania 1, intestinal atony 1.

(*k*) Surgical Conditions.—Abscess of liver 4, fractured rib 3, abscess of back 2, necrosis of rib 2 (empyemas), perforation of diaphragm 1, perforation of chest-wall 1, tumor of lung 1.

Urine. The results of the examination of urine are of interest. Urine records were found in the histories of 470 cases. The examinations after 1898 were conducted in the Ayer Clinical Laboratory of the hospital, under the supervision of the Director; and from that time, at least, have been systematic and accurate.

It appears that acute nephritis is not more frequent in empyema than in other pleurisies, actually as 1 in 6 is to 1 in 6½. A particular investigation of the urine of admitted alcoholics revealed some surprising findings. In a general way it may be stated that their renal condition was either exceedingly bad or else entirely normal, and, surprising as it may seem, it was usually the latter. There was practically no intermediate condition. In two-thirds of the alcoholics the urine was entirely negative, whereas in only seven-sixteenths, or less than one-half of the non-alcoholics, was this the case.

Even allowing for the perhaps numerous alcoholics who were improperly classified, owing to concealment of their habits, still the proportions are considerably at variance with the accepted views on the relation of alcoholism and nephritis. Of the 40 patients who died, 8 gave an alcoholic history, which is exactly the same death rate for alcoholics and non-alcoholics. In pneumonia, as Norris has shown, the drunkards fare much worse.

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[9] 1 single, 1 double.

Acute nephritis was found in 77 cases and chronic nephritis in only 29 of the 500. In 16 cases casts were found, but not albumin; generally only hyaline casts. A few contained granular casts, and may be added to the list of chronic nephritis.

In but 14 cases were uric-acid crystals found, whereas calcium oxalate was found nearly as often, that is, in 10 cases.

Bile was found in 7 cases, nearly all complicated by cholecystitis; hemorrhagic nephritis occurred but thrice.

Cases in which urine was entirely negative	231	
Alcoholic cases in which urine was entirely negative	66	(out of 102 total).
Cases with only a trace of albumin and no casts	91	117 irritation.
Cases with only a trace of albumin and only hyaline casts.	26	
Cases with acute nephritis (albumin and casts)		16 empyemas, 61 not empyemas.
Cases with chronic nephritis (albumin and casts)		4 empyemas, 25 not empyemas.
Cases with no albumin, but showing casts	16	

	Cases.
Calcium oxalate	10
Uric acid	14
Bile	7
Hemorrhagic nephritis	3

Blood. The blood counts are by no means numerous, and apart from the leukocyte counts show little interest. As regards the erythrocytes, counts were recorded in but 21 instances, and these counts were made in complicated cases, in which other diagnostic points were involved. In 5 of these cases the counts were over 5,000,000, and in one was 6,000,000 after aspiration. In but 1 case was the count less than 3,000,000—a case complicated by ulcerative endocarditis.

The hemoglobin was estimated in but 27 cases, and the results are very variable. It was over 90 per cent. in 5 cases, 100 per cent. in 1 case, and generally speaking was uniformly high. Twice only, and then in complicated cases, was it below 55 per cent. Disproportionate oligochromemia was noted twice; in 1 case with over 5,000,000 red cells, the hemoglobin was but 45 per cent. In the tuberculous cases the leukocyte counts were the lowest, only in 2 instances (both cases of empyema) exceeding 10,000. In empyema (not tuberculous) the count was generally high, though in 1 instance it was below 6000 and in 3 others below 10,000.

In complicated cases the leukocyte counts usually rose considerably above the counts in the simple serous pleurisies, but in only 2 cases were over 50,000, both of which had croupous pneumonia. The Widal reaction as resorted to occasionally was disappointing. In 10 cases with typhoid-like condition, but not typhoid fever, the reaction was negative; but in 9 cases, also undoubtedly not typhoid fever, 4 positive and 5 highly suggestive reactions were obtained, and no history of recent typhoid infection could be obtained.

In 3 cases of miliary tuberculosis the Widal reaction was suggestive twice and negative once. Blood cultures were resorted to four times; in one instance a pure culture of pneumococcus was obtained, which was also found in all cultures postmortem. Malarial organisms were found twice in fresh blood preparations.

LEUKOCYTE COUNTS.

	Less than 6,000.	6,000–10,000.	10,000–20,000.	20,000–30,000.	30,000–40,000.	Over 50,000.
In empyema (non-phthisical) . .	1	3	19	10	6	1[10]
In phthisical cases of all kinds . .	6	4	2[11]			
In non-complicated pleurisies . .	12	20	22	2		
In complicated pleurisies	5	16	25	10		1[10]

SPUTUM. Many examinations of the sputum were made, but the results may be briefly tabulated as follows: Tubercle bacilli were found in 3 empyemas, and in 11 other cases. Pneumocci were found in 6 empyemas, and in 18 other cases.

THE EFFUSIONS. In almost all the serous cases the specific gravity was between 1015 and 1025. The amount of albumin present was more variable, but usually ranged between 15 and 40 grams per liter. In 2 cases the albumin was extremely high; once 70 and once 80 grams per liter. The bacteriological examination of the exudates was as follows:

	Tubercle bacillus.	Pneumococcus.	Streptococcus.	Bac. col. communis.	Sterile.
In the examination of aspirated pus .	1	24	10	0	11
In the examination of aspirated serum .	2	13	1	3	50

	1010–1015.	1015–1020.	1020–1025.	1025–1030.
Specific gravity of effusion	3	15	24	1

	Under 1000.	1010–1015.	1015–1020.	1020–1030.	1030–1040.	1040–1050.	1070.	1080
Albumin, grams per liter .	2	4	10	13	13	5	1	1

In 1, effusion sugar was found; in 1, liver cells and tyrosin bundles; in 1, liver cells and hematin and bilirubin crystals. In 1, empyema cholesterin crystals were found. In 2, empyemas following hepatic abscess, examinations were made for amœba with negative results. In the single case of tumor, blood cells and endothelial cells were numerous.

CYTODIAGNOSIS AND INOSCOPY. A series of 22 cases of pleural effusion was studied by Dr. G. C. Robinson, resident pathologist at the Pennsylvania Hospital, with the following results: In 19 cases the small mononuclears were from 95 to 100 per cent. of the count. Three showed a polymorphonuclear excess (1 seropurulent). Cultures were negative in all of the 9 cases examined.

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[10] Pneumonic.

[11] Both complicated.

Inoscopy after Jousset's method, in 15 cases, showed tubercle bacilli in 2 cases (1 in pericardial fluid). In 1 case that showed tubercle bacilli in the sputum there was an effusion with polymorphonuclear excess in the count.

Physical Signs and Symptoms. Bulging of the chest or obliteration of the interspaces is noted as occurring in 15 empyemas and 50 serous effusions. Movable dulness is noted but 40 times. Egophony was noted in 5 empyemas and 48 serous (usually small) effusions. Pectoriloquy occurred in 8 cases, bronchophony in 3, amphoric breathing in 4, bell tympany in 5, metallic tinkling in 3, and succussion splash in 2 cases. Skodaic resonance is mentioned in but 4 cases, probably because it is so frequently found that it is not considered worthy of record. Distant bronchial breathing over an effusion is almost as frequent as is the absence of breath sounds.

A difference in the mensuration of the two sides of the chest is mentioned but 15 times; twice it was said to be the same on both sides. The liver was found displaced appreciably below the costal border 68 times, in all but 9 of which effusion was right-sided (5 times with a massive left effusion and twice with the left-sided effusion complicated by a cholecystitis). The spleen was found displaced appreciably below the costal border 23 times, in all but 2 of which effusion was left-sided (malaria, typhoid, etc., omitted).

Jaundice was noted in 5 cases; herpes in 5 cases with pneumonia, and in 8 cases without pneumonia. Hemoptysis occurred in 3 cases. A putrid odor of the breath was found in all the 4 cases of abscess of the lung.

Œdema of the chest occurred 8 times. Pulsation of the right side was observed in a left-sided effusion where pericardial adhesions were present. Extreme abdominal distention was noted in 1 case. Marked tenderness to light pressure or percussion over the affected side was present in 9 non-purulent cases. Persistence of friction was noted in 1 case eleven weeks, in another eight weeks, and in another five weeks.

Anisocoria was noted in 7 cases, in one of which, complicated by locomotor ataxia, the Argyll-Robertson pupil was observed. In the other 6 the pupil was larger on the affected side five times and smaller once.

Subcutaneous emphysema occurred after aspiration in 1 case, but rapidly disappeared without bad effect. Delirium was noted late in the course of the disease in non-alcoholics four times.

Meningismus was present in one child, and hemicrania of the affected side was also once noted. In 1 case of double pleurisy with marked physical signs there were practically no symptoms. Another case had a typical abdominal onset, no pulmonary symptoms, and an effusion was found in the course of a routine examination of the chest. The tuberculin test employed three times was twice positive and once negative. Skiagrams in 3 cases located missing drainage tubes in the pleural cavity, and in 1 case a grooved director, which was recovered from the chest a week after the original operation.

A few anomalies of pulse and respiration are worth recording. In 1 massive effusion the highest respiration-rate was 24. In another with both friction and effusion it was 20. In 1 case with a temperature of 105° respirations were 28, and in 1 case with a subnormal temperature the respirations were 44. In 1 case with large effusions the highest pulse-rate was 64. In another with respirations 36, the pulse was but 72; and in another with respirations 40, the pulse was 80. In still another case with the respirations 54, the pulse was but 104, and later on in convalescence went as low as 54. In another case with very great cardiac displacement the pulse was never over 96, and it fell to 48 after aspiration as the heart returned to its normal position.

Results of Treatment. It is impossible in this paper to review the medicinal treatment of such a series of cases. The number and combinations of various drugs employed defies analysis, but we may note that strapping of the chest is always reported as giving prompt and marked relief of pain, cough, and dyspnœa.

We may, however, review and contrast those cases which received only medical treatment with those in which aspiration was in addition performed, and we may also see what was accomplished by operation in cases of empyema. Furthermore, we may learn to what extent early treatment influenced the course and termination of the disease, and finally we may inquire into the causes which brought to a fatal termination 40 of the cases in which medical and surgical resources were exhausted in vain.

Again, we have for convenience divided the cases into those of short (first week), medium (second week), and long (over two weeks) duration, and will consider separately the serous effusions, the empyemas, and those cases in which no effusions occurred.

Three hundred and seventy patients were discharged as cured, and 90 others left the Hospital, of whom all but 4 were improved at the time of their discharge; 38 of these patients left against the advice of the physicians in charge, and the other 52 represent mostly phthisical patients and cases of empyema which still had sinuses that were referred to the out-patient department in order to make place in the wards for more acute and urgent cases. None of these cases are included in the results, though they were nearly all practically cured at the time of their discharge.

Early treatment shows remarkable results, as evidenced by a study of the 78 cases which were admitted in the first three days of the disease. Of these, 40 never developed effusion, 18 a slight basal effusion, and 8 an effusion of one-third of the chest; none of these 66 cases required aspiration. But 2 developed large effusions, and the other 10 were empyemas (mostly pneumonic).

Of the 78 cases admitted in less than 4 days from onset:

No effusion	40	None required aspiration.
Base	18	
One-third	8	
Large effusion	2	
Empyema	10,	all pneumonic but 1 trauma and 1 abscess of liver.

CURED.

	1 wk.	2 wks.	3 wks.	4 wks.	5 wks.	6 wks.	2 mos.	3 mos.	4 mos.	5 mos.	6 mos.	1 yr.	
NO EFFUSION.													
Short duration	29	23	7	8	2	.	..	..	..	..	..	..	69
Med. duration	4	6	1	2	0	1[12]	0	1[12]	..	..	..	..	15
Long duration	3	1	0	1	..	..	..	..	..	..	..	..	5
SERUM.													
Short duration	10	21	16	12	6	7	2	0	1	..	..	..	75
Med. duration	5	22	27	12	12	5	0	2	1	..	..	..	86
Long duration	12	10	16	11	11	4	2	4	0	..	..	..	70
PUS.													
Short duration	....	..	..	1	0	1	2	9	5	0	1	1[13]	20
Med. duration	....	..	..	0	0	1	0	1	2	2	..	..	6
Long duration	....	..	..	3	2	3	4	6	4	2	..	..	24
Total	63	83	67	50	33	22	10	23	13	4	1	1	370
DEATHS	16	7	4	2	3	1	2	3	1	0	1	..	40

DIED	Pus 21.	Left against advice, improved	34
	Serum 16.	Left against advice, not improved	4
	No effusions 3.	Discharged improved, including phthisis and empyema still with sinuses	52
		Total	500

Of the dry pleurisies we find the great majority cured in the first two weeks (36 in the first week); and the average duration before cure was fifteen days for the entire series, including complicated cases; 77 per cent. of this series were admitted in the first week of disease.

Of the 231 serous effusions the average was twenty-five days, and half the cases were cured in the second and third weeks.

The time of admission is very evenly divided between the short, medium, and long periods, but is slightly in favor of the medium.

Of 320 non-purulent cases all but 13 were cured in less than two months.

With the empyemas we find a very different state of affairs. Almost all the cases were admitted early (generally with pneumonia, developing the empyema in the hospital), or else after the effusion had been established for some weeks, and of these two classes there are almost an equal number, only 6 of the 50 cases being admitted in the second week.

Vastly different also is the table of recoveries and duration of treatment. No patient was cured in less than four weeks, and the average is ninety days, or exactly three months, in which column are found-one-third of the cases. On the other hand, all but 6 patients were cured in four months or less, and these were severely complicated.

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[12] Complicated.

[13] Child with numerous complications.

TAPPING VS. NON-TAPPING IN SEROUS EFFUSION. In comparing the results achieved in which mechanical interference was employed with those in which no such procedure was attempted, it soon became obvious that only uncomplicated cases could be fairly contrasted. The complications varied so greatly in severity, in some cases lengthening the course of the disease by many weeks, and in others having little or no such influence, that deductions from these cases would be decidedly misleading.

In the accompanying table, which details all the complicated and uncomplicated cases, it will at once be seen how variable are the results in the cases with complications, for in these cases it appears that those not tapped recovered much the sooner. The facts are, that the complications were much severer in those tapped, and possibly for that very reason the aspiration became imperative. Only cases discharged as cured are considered, and slight effusions at the base are not included, as in no cases were such aspirated:

COMPARISON OF EFFUSIONS TAPPED AND NOT TAPPED.

TAPPED.

UNCOMPLICATED.	1 wk.	2 wks.	3 wks.	4 wks.	5 wks.	6 wks.	2 mos.	3 mos.	4 mos.	Average in days.
One-third	4	6	3	2	..	..	..	..	..	15.4
Two-thirds	5	8	19	4	2	0	1	..	..	20.1
Massive	2	6	2	8	6	1	..	..	..	24.7
Mean average										20.6
COMPLICATED.										
One-third	1	0	2	4	1	3[14]	1[14]	..	..	31.5
Two-thirds	..	1	0	2	2	2	1	3	..	50.4
Massive	0	2[2]	2	3	4	2[14]	2	..	..	32.7

NOT TAPPED.

UNCOMPLICATED.	1 wk.	2 wks.	3 wks.	4 wks.	5 wks.	6 wks.	2 mos.	3 mos.	4 mos.	Average in days.
One-third	2	5	2	6	1	1	..	..	..	21.8
Two-thirds	0	2	8	2	2	1	..	..	..	24.3
Massive	0	0	1	0	2	1	0	1	1	55.0
Mean average										28.0
COMPLICATED.										
One-third	2	2	7	1	2	1	1	..	..	24.0
Two-thirds	0	0	0	0	3	1	..	..	..	37.0
Massive	..	..	..	..	..	..	..	..	..	....

To recapitulate, it appears that, taken as a series, uncomplicated cases of serous effusions aspirated recovered in three weeks, whereas another week was necessary to effect a cure in the non-aspirated. Strangely enough, the contrast is more marked in the small effusions than in those of moderate size.

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[14] Includes double pleurisies with other complications.

In the massive effusions the contrast is mostly afforded by those patients who refused aspiration, and the encouraging average of 24.7 days in the aspirated as against 55 days in the other group makes further comment unnecessary regarding aspiration in such cases.

It is but fair to say, however, that in hospital practice aspiration is performed under the most favorable conditions possible for asepsis, and with the best chances for combating any hitch or emergency that may arise in the proceeding.

In private practice, on the other hand, I am of the opinion, taking all the factors into consideration, that in an uncomplicated serous effusion not exceeding what is here termed "one-third" of the chest, aspiration should at least be delayed until it becomes evident that absorption will not occur naturally in two weeks, as it did quite frequently in the non-aspirated small effusions in this series.

Treatment of Empyema. These statistics emphasize the fact that in resection alone is there safety. Even then in 75 resected cases there were 13 deaths. The mortality is high at all times, 22 per cent. for our entire series, but only 17 per cent. for those resected, as against 40 per cent. for those not resected. In those cases in which simple incision and drainage was performed the mortality was 27 per cent., and in those not drained 5 out of 9 died; of the other 4, only 1 was cured at the time of leaving the hospital, which they did notwithstanding earnest protest.

Mortality and Causes of Death. No uncomplicated case of non-purulent pleurisy died. Three cases of pleurisy without effusion succumbed, but 2 of them occurred late in the course of typhoid fever, and the third was an alcoholic patient, who developed delirium tremens; death occurred in 16 of the 315 serous effusions (5 per cent.). The mortality for the entire series (40) is exactly 8 per cent., which for hospital cases is not unduly high. A majority of the deaths occurred early (16 in the first week after admission), and consisted mostly of those cases complicated by pneumonia and acute nephritis, and of empyemas moribund from septicemia.

The causes of death were as follows:

IN EMPYEMA.

Septicemia	7	(1 with pericarditis)
Pneumonia	3	(1 with nephritis)
Abscess of lung	2	
Phthisis	2	(1 with malaria)
Nephritis	2	(1 with myocarditis)
Endocarditis	1	(with tuberculosis)
Pericarditis	1	(with peritonitis)
Abscess of liver	1	(with perforation of diaphragm)
Exhaustion after operation	2	
Total	21	

IN NON-PURULENT CASES.

Pneumonia, croupous	3
Pneumonia, senile	1
Pneumonia and alcoholism	1
Pneumonia and phthisis	1
Miliary tuberculosis	1
Miliary tuberculosis and abscess of liver	1
Miliary tuberculosis and pericarditis	1
Typhoid fever and nephritis	2
Acute nephritis	1
Pericarditis and femoral thrombosis	1
Pneumothorax and tumor of lung	1
Abscess of liver	1
Cirrhosis of liver	1
Acute mania and shock	1
Ruptured aneurysm	1
Septicemia (with purpura)	1
Total	19

I venture to think that this table accounts fully for as high a death rate as 8 per cent. In only those 2 patients who died of exhaustion after operation might a different result have been expected, and had not operation been performed the end would have surely been fatal.

Autopsy Notes. Autopsies were performed in 22 cases, of which 11 were empyemas. In the non-purulent cases pulmonary tuberculosis, acute, subacute, or chronic, or a general miliary tuberculosis was found in 9 cases. In 2 of them there was tuberculous pericarditis, and in 2 the effusion was hemorrhagic. Carcinosis of the pleura was found in 1 case. Among the empyemas, but 1 had pulmonary tuberculosis. Pericarditis was found three times (twice with right-sided empyemas). Five loculated empyemas occurred. Evidently in this disease there is attempt to wall off the pus, but in 2 cases it found an outlet rupturing into the bronchi; in 1 also infiltrating the axillary tissue, and in 1 the chest wall. Acute endocarditis was found in 1 case. In 1 case of typhoid fever a double empyema was present. Abscess of the liver was found twice, and femoral thrombosis also twice.

Conclusions. 1. Pleurisy occurs at all ages, but most frequently from twenty to thirty.

2. Pleurisy is most frequent in the spring and least in the fall.

3. Pleurisy affects children under fifteen with equal frequency for boys and girls, but is four times as frequent in men as in women.

4. The colored race is particularly susceptible.

5. Occupation seems to have a decided predisposing effect.

6. Croupous pneumonia is the most frequent cause of empyema, and bronchitis, or simple exposure to dampness and cold, seems to be more frequently the exciting cause of pleurisy than tuberculosis or rheumatism (as far as clinical evidence is found).

7. Complications are frequent in hospital practice.

8. Aspiration shortens appreciably the course of the disease, and usually requires to be performed but once to effect a cure.

9. Examination of the blood is but moderately helpful and of doubtful value, as are also cytodiagnosis and inoscopy.

10. Empyema is always dangerous, and its natural course almost invariably fatal. Resection and free drainage is the proper treatment.

THE RELATION OF THE KIDNEYS TO GASTRO-ENTEROLOGY.

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In a broad way, renal diseases may be classified as follows: (1) Neoplasms, of interest mainly to surgeons and pathologists. (2) Local pathological lesions, such as infarcts, scarcely diagnosticable. (3) Parasitic disease: (*a*) gross animal parisitism, rare in this climate; and (*b*) bacterial infections, septic, gonococcic (ascending), tuberculous, exanthematous (included as a matter of convenience), and other acute infections. (4) Traumatisms. (5) Necrosis. (6) Displacements. (7) Calculus disease and certain forms of urinary obstruction. (8) Inflammations or degenerations, involving the kidneys generally (Bright's disease, etc.). (9) Functional disturbances: (*a*) motor, impossible, except in an indirect sense of involvement of the vessels in ischemia and congestion; (*b*) secretory, excessive, deficient, or perverted; and (*c*) sensory, neuralgia (also, true neuritis of the kidney).

While almost any kind of disease of any part of the body may occasionally produce, or be accompanied by, some definite disturbance of digestion or call for special attention to diet and metabolism, the following partial classification of renal conditions is suggested as pertinent to our consideration: (1) Mechanical influences: (*a*) movable kidney, and (*b*) calculus disease and urinary obstruction. (2) Chemical and allied influences: (*a*) acute nephritis; (*b*) chronic nephritis; (*c*) renal paresis and paralysis; (*d*) polyuria; and (*e*) urinary abnormalities secondary to digestive and metabolic disturbances, without strict renal disease.

The relatively large number of cases of movable kidney encountered in digestive practice must be significant. Even gynecological series do not usually show more than 10 per cent. Ralph C. Larrabee[1] found 112 cases among 272 women—41.5 per cent. Wolkow and Delitzin[2] found 46 per cent. Others have reported an incidence of 60 to 80 per cent. The personal equation must enter into the matter. I have considered as movable only those cases which showed an unmistakable, palpable change of position beyond anything that could be ascribed to elasticity of the abdominal wall. In my cases no significant association with any particular digestive disease has been demonstrable, certainly not with inflammation of the appendix, nor with genuine gastroptosis. I have found altogether, in many hundred cases, only two of hepatoptosis, and have not been convinced of a typical general splanchnoptosis. But all of my results, in any line of investigation, are counted as negative unless unmistakable abnormality has been found on rigid objective study. If one counts every case of atonic dilatation of the stomach as gastroptosis, every case of flabby abdomen as enteroptosis, and the combination of two or three ptoses as a general splanchnoptosis, it is easy to make up comparatively large series. It should be distinctly understood that this statement is made without prejudice and without the least idea of reflecting on the much larger series of several competent observers, abroad and in America. Differences of clientele may easily affect the statistics.

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[1] Boston Med. and Surg. Jour., November 26, 1903.

[2] Die Wanderniere, 1899.

STATISTICS OF MOVABLE KIDNEY FROM RECENT PRIVATE PRACTICE, NOT INCLUDING THE PRESENT FISCAL YEAR.[3]

	No. in series.	No. of cases.	Percentage of incidence.
Males living	321	15	4+ per cent.
Females living	314	83	26+ "
Total living	635	98	15+ "
Males dead	120	4	3+ "
Females dead	82	9	11— "
Total dead	202	13	6+ "
Total males.	441	19	4+ "
Total females	396	92	23+ "
Total patients	837	111	13+ "

ETIOLOGY. I have found very few cases of movable kidney among two or three hundred children from early infancy to puberty, and, so far as my experience goes, the following classic causes are not operative: loss of weight, frequent or even any pregnancy, corsets, heavy lifting, straining, traumatisms, exhausting sicknesses, etc. I have made out no significant hereditary or familial tendency, although it is common enough to find it in two cases in a family, in the same or different generations. It is not significantly associated with hernia and other displacements of viscera, although such coincidences are, of course, quite frequent. The essential cause seems to be failure of abdominal development at puberty, when the pelvis enlarges.

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[3] Not counting series examined in asylums and hospitals especially to find movable kidneys, I have careful records of 170 cases of this condition, the recognition of which in this country has been comparatively recent. A larger series, from the records of Dr. C. D. Aaron, will be discussed with these in a subsequent paper.

Nephropexy often fails signally and there is, perhaps, danger in cutting, even with needle wounds, excretory tubules. Curiously enough, I have found movable kidney especially frequent in physicians' wives. In many cases the gastric symptoms which lead the patient to consult a digestive specialist seem purely reflex, the physical and chemical examination of the stomach resulting negatively. None of my nearly 300 cases has had Dietl's crises. One was supposed to have had but a careful examination and inquiry into the history failed to support the diagnosis. Without questioning the authenticity of reports of this condition, it certainly must be extremely rare, and one is tempted to suggest that surgeons may make the diagnosis without adequate ground, and to draw a parallel with the unprecedented frequency of gastric ulcer in surgical series, as contrasted with medical and postmortem statistics.

Renal Calculi and Urinary Obstruction. These conditions, though often marked by vomiting and constipation, the latter even amounting to obstipation and due to reflex spasm, do not directly come under our observation for acute attacks. The prophylaxis of calculus disease in general, however, is a very important function of the digestive specialist. Routine examinations of the urine in patients subject to urinary calculi should always include an investigation of the acidity, a matter which will be discussed in another article.

The differential diagnosis of renal calculus is sometimes exceedingly difficult. Other forms of obstruction, biliary lithiasis or inflammation, scolecitis (appendicitis), colic of the appendix, inflammatory conditions of the bowel, especially limited as to the ceco-appendix, sigmoid, etc., gastric ulcer, pancreatic disease, and, in cases of suspected hydronephrosis or pyonephrosis, any tumor, splenic or otherwise, may be confused.

J. B. G. (No. 56, of 1904–05) had an acutely painful attack which his attendant considered biliary colic. An excellent general practitioner, called in consultation, unhesitatingly diagnosed renal colic, although, in justice to him, it should be said that he had somehow obtained a false idea of certain conditions, including hematuria, which had never been actually demonstrated. The patient was subsequently turned over to me, there being some fever and leukocytosis, but only average urinary acidity. My diagnosis lay between biliary lithiasis and some septic involvement of the appendix. Operation was refused. Some months later another attack led to operation and a gangrenous appendix was found. Death followed. In other cases it has been found impossible to distinguish between renal colic and sigmoiditis or left-sided colitis, at least without following the case past the acute attack.

Nephritis. Acute nephritis occurs mainly in connection with infectious fevers or such exposure to chill and metabolic or ordinary saprophytic toxins as withdraws it from our experience. In 713 serial cases I saw it only once in consultation. Nephritis or even marked congestion does not occur so frequently in typhoid as in the fevers outside of our specialty, and, if typhoid patients are adequately fed, on cereals as well as milk and beef-juice, and some pains are taken to prevent intestinal decomposition, renal complications will be exceedingly rare.

Chronic nephritis is quite infrequent in digestive practice, especially when we consider the frequency of hepatic sclerosis and the history of alcoholic indulgence, drugging with arsenic and various other irritants, and the formation of toxic products in the alimentary canal or in general metabolic processes.

Among 517 living patients, 4 showed chronic diffuse nephritis, 1 multiple visceral lesion (A. O., M. R., T. R., anemia, chronic nephritis, some cerebral lesion producing temporary aphasia, apparent recovery reported by attendant), 2 persistent albuminuria without casts (one of the last, orthostatic albuminuria). Among 196 dead subjects, 8 showed chronic or subacute tubular nephritis, the diagnosis as to the dividing line being difficult in such cases, which are under observation for a short time previous to death; 12 showed multiple visceral lesion, although in some there was merely albuminuria and no casts, so that the kidney may be considered as only congested; 1 showed albumin and albumose, the nature of the case being puzzling (P. S., No. 60, 1898–9); 1 showed renal insufficiency and auto-intoxication without genuine Bright's disease. Altogether 713 cases included only 29 that could be considered as distinctly renal lesions, an incidence of 4 per cent. for the whole series, about 1.5 per cent. for the surviving cases and 11 per cent. for the fatal cases. On the other hand there were several of extreme cardiac disease, some with hepatic sclerosis of marked type, atrophic or hypertrophic, abdominal or general dropsy and ultimately pulmonary œdema in which the kidneys maintained themselves to the end. In one case of multiple visceral lesion there was a terminal bronchopneumonia, general dropsy, and a decided right-sided anasarca appeared a few days before death. The pneumonic condition was more marked in the right lung, and there was found postmortem more fluid in the right pleural cavity, but the sharp unilaterality of the dropsy could not be explained by posture, nor by anything observed before death or at the necropsy. It was considered to be due to a unilateral angioparalysis, but this only makes the ultimate explanation more difficult.

With reference to the diagnosis of renal lesions from the urine, allusion may be made to the classification of urinary casts by Simon and others, to a recent series of articles by Emerson, and to Cabot's valuable skeptic comparison of clinical and postmortem results, published last year. The conflict between the older and some of the more modern views may be harmonized to some degree, if we regard the matter with the following practical qualifications:

1. Minor lesions of the kidneys, demonstrable by microscopic examination of sections, may escape recognition on account of the dilution of diseased by healthy urine.

2. On the other hand, foci of inflammation or degeneration may produce albuminuria, casts, etc., and ultimately recover or be filled in by scar tissue.

3. Even quite extensively diseased kidneys may occasionally excrete urine that eludes an ordinary clinical search for abnormalities.

4. Normal urine contains a trace of proteid, and any normal organ must shed more or less epithelium or be subject to conditions practically inflammatory, but of minute area.

5. Under strain any kidney will pass albumin and casts. Whether the condition should technically be considered nephritis or not is not of practical importance.

6. Unless foreign albumin is introduced directly into the circulation or is absorbed from excessive amounts ingested, as after a diet of raw eggs, there is no condition analogous to hyperglycemia. Hence there can be no such thing as physiological albuminuria, excepting as stated. It is exceedingly doubtful whether an albuminuria can be functional.

7. On the other hand, unless an albuminuria has an obvious source below the kidney, casts are rarely lacking, and a clinical albuminuria, not readily attributable to the lower passages, is almost always an indication of true renal congestion or nephritis.

8. Unilateral renal disease without obvious cause and indications is rare.

9. For clinical purposes albuminuria can be conveniently estimated by boiling and centrifuging, the moist precipitate exaggerating the actual percentage of albumin five or six times. With due allowance for temporary fluctuations, bulk of urine, and the particular kind of renal lesion present, the amount of albumin is an approximate measure of the grade of the lesion, although it would be obviously absurd to draw prognoses directly from the centrifuge readings.

10. The ordinary clinical observer (a personal confession is here included) cannot always make a sharp classification of the different kinds of casts nor always distinguish between true and false casts. Whether experts can do so or not need not be discussed. However, all studies, in which the attempt has been made to identify every cast seen and to make a diagnosis from the urine alone, at one or a few examinations covering a brief period, show that it is impossible to connect a microscopic field with a definite pathological lesion.

11. However, by considering both clinical, chemical, and microscopic examinations, and by taking the general conspectus of the microscopic examinations covering a reasonable period, I believe that it is possible to distinguish pretty sharply renal congestion, interstitial and tubular nephritis, and, of course, amyloid disease,

hemorrhage, etc., and to gain a fair idea as to the pathological stage of the lesion.

12. Nitrometric estimations of urea are really rough tests for total non-proteid nitrogen. They certainly include uric acid and ammonium salts.

13. The various quantitative tests for urea, ammonia (by liberating ammonia gas and titrating a standard acid solution used to absorb it), uric acid (by R. Harvey Cooke's test), and salines (by titration or centrifuge) are of considerable value in keeping track of metabolism, although they do not yield diagnostic information in the sense of enabling the application of names to renal conditions.

It would require too much space to consider the ways in which renal and strictly alimentary conditions react upon one another. Croftan has emphasized a point not altogether novel, that uremia is not due in a direct, crude sense to the retention of urinary waste. Bouchard's statements that urinary waste kills if accumulated for twenty-four hours, hepatic waste in nine hours, are true only in an illustrative sense. It is doubtful whether, for practical clinical purposes, we can distinguish these sharply. At any rate, their production certainly falls as does the elimination, although not *pari passu*. So, too, in chronic disease of the emunctories, we must assume an increased tolerance to the poisons.

J. J. S. (No. 99, of 1905–06) has amblyopia, diagnosticated as due to albuminuric retinitis. He also manifests the general toxemia conected with hepatic sclerosis and chronic diffuse nephritis, yet forn long periods his urine is qualitatively normal. It has never yielded as much as 1 per cent. of moist precipitate and the elimination of urea, uric acid, and salines is within normal limits. It is scarcely necessary to state that the real toxic matter has not been isolated nor that, if there is any one characteristic toxin, it must be something present in very small amounts.

H. W. G. (No. 1050, of 1895–96) showed mitral regurgitation, albumin, and casts for several years, and the case was considered one of true chronic diffuse nephritis. In 1899, during an attack of cholera morbus, he was anuric for nearly a week, without developing uremic symptoms. Indeed, there is a paradox of pretty general applicability, that anuria, even existing for a week or two, does not produce uremia. In 1900 the patient had a severe attack of phlegmonous erysipelas and was thought to be dying. Since then several urinary examinations, at long intervals, have failed to show any urinary abnormality. Did the erysipelatous toxemia cure the nephritis?

Renal albuminuria without casts is a subject that has occupied much space in the literature of the last few years. Objection has already been made to the terms physiological and functional. Cyclic is also an objectionable term, as in the case of cyclic vomiting. The fact that a condition appears and disappears, even at fairly regular intervals, does not warrant the term cyclic, unless there is a definite and regular dependence upon some inevitably and uniformly recurring circumstance. In one of my cases the albuminuria was distinctly orthostatic, depending upon the vocation of conductor on a street car, the urine passed after rest in bed being free from albumin. Most of my cases have occurred after adolescence. As casts are occasionally found in normal urine, especially after strain and when large volumes are centrifuged, an occasional cast does not indicate a separation of the case from this clinical group. Orville A. Kennedy[4] reports a case at first showing only cylindroids, but after a few years genuine tube casts, and the general consensus of opinion seems to be that these forms of albuminuria are essentially nephritic, though the lesion is mild and even curable.

A very practical consideration is the extent to which the stomach in nephritis acts vicariously for the kidney. We have probably all of us laughed at physicians who have called the jejune vomitus in Bright's disease vicarious urine. Doubtless the expression has been due to an entirely superficial appearance. Yet we must remember that such liquid necessarily contains the urinary salts and certain organic compounds and even notable quantities of urea. At any rate, lavage seems to have not only a local effect, but really to aid in the elimination of toxins.

DELIRIUM TREMENS. This symptom-complex is mentioned in this connection because cases of auto-intoxication with sclerotic livers and badly congested or diseased kidneys have so frequently presented delirium that, from the standpoint of the general practitioner, would be properly called delirium tremens. Indeed, the difference of phraseology may be considered as a divergence of diagnosis. It has long since been pointed out that delirium tremens is not drunkenness and that alcohol often relieves the condition. Without any considerable opportunity to study ordinary cases of delirium tremens, I would like to raise the question whether there is anything essential in the symptom-complex that cannot be caused in total abstainers, and whether, considered as a group, all cases of so-called delirium tremens cannot be reduced to auto-intoxication with waste matters commonly eliminated by the liver or kidney or both. No one disease of either organ can be considered necessary. One may seek and often find hepatic sclerosis and chronic nephritis. But, as the late H. F. Formad pointed out, the really characteristic drunkard's kidney is the pig-backed kidney with chronic congestion. Then, too, delirium tremens may occur before hepatic sclerosis has had time to develop.

One of my latest fatal cases (No. 10, of 1896–97), aged forty-three years, was the son of a man who died of typical atrophic hepatic sclerosis. Both had been rather liberal users of alcoholics, though the father presented no true alcoholism. The son had had several alcoholic attacks, including such lesions as pericarditis and lobular pneumonia and his liver was quite contracted. In the last attack it was much enlarged, but speedily contracted to its permanent minimum under calomel. He also had right lobar pneumonia. The urine could not be saved until just before death, when it was found to contain 7 per cent. of sugar, but neither albumin nor casts. There had been no previous symptoms of diabetes and the urinary excretion, though liberal, so far as could be judged, was not excessive and there was no polydipsia. Still, so large a percentage of sugar, after several days of very light diet, almost justifies the term diabetes. Necropsy could not be obtained.

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[4] AMER. JOUR. MED. SCI., March, 1906.

Curiously enough, in about fifty cases of delirium tremens, I have never noted hallucinations of snakes; rats, horses, dogs, cats, and human beings have been the most frequent hallucinations. The last patient mentioned saw playing cards on the ceiling. Sometimes the hallucinations have been general, sometimes individual. With rats, lions, tigers, etc., the patient would simply see such animals. Human beings are usually individualized. Domestic animals are sometimes seen as individuals, sometimes merely as animals. In one case, the patient, himself an Irishman, described his hallucinations as of red-headed Irishmen, but apparently recognized none as individuals. Fear may or may not be present and the fear may be a logical realization of danger, as in the case of lions and tigers, savage horses and dogs, or a morbid horror as in the case of rats and mice. Most human beings have more or less ophiophobia, so that if snakes are seen at all there is likely to be mental terror. As a rule, however, terror seems to be lacking and the visions may even be amusing or pleasurable. There is even a story about a zoölogist who experienced scientific pleasure in analyzing the snakes that he saw. With the popular and even professional conception that delirium tremens patients see snakes it is a wonder that suggestion does not lead to the much more frequent presence of this hallucination.

According to one theory, the vision of snakes is not an hallucination, but an illusion due to the congestion of the tortuous and snake-like retinal vessels. I will leave to a more expert physiologist the explanation of how even a congestion of these vessels could lead to sensory impressions of objects of similar contour. That the explanation does not hold is evident from the actual rarity of ophioscopia and from the fact that pasting paper over holes through which the patient saw the snakes enter, or driving them out of the room, or otherwise humoring the patient's delusions, sometimes causes the hallucination to vanish. A much more rational objective explanation is that the blood corpuscles, whose shadows can actually and easily be seen by anyone, form the basis of a visual illusion.

As in other manifestations of auto-intoxication, the prime points in the treatment of delirium tremens are to stimulate, with digitalis, strychnine, hot saline rectal injections, external heat, etc., to purge and stimulate the kidneys and skin, and to avoid an overdose of any depressant by using a succession of nerve sedatives, rather than any one to excess. Morphine should be used only with the utmost caution.

Multiple Visceral Lesion is a term that I introduced several years ago, to cover cases in which the heart, lungs, kidneys, liver, spleen, and alimentary canal are mutually involved in chemical and mechanical processes, through the circulation. Sometimes the heart, sometimes the kidneys, sometimes the liver, seems to be the primary locus, but various vicious circles become established. Excepting as dyspeptic conditions or the ingesta directly affect the liver, the alimentary canal is not primarily involved, but eventually becomes catarrhal on account of portal obstruction, and there may be capillary oozing, or the formation of varicose internal ulcers, or massive hemorrhages, due to this interference with return circulation. Pick's frosted liver may exist in such conditions. The pulmonary condition is usually terminal, being a combination of catarrh and œdema.

A very important, and, to me, inexplicable fact is that in renal disease there may be a pretty definite tendency to water-logging with mechanical crippling of the circulation and respiration, or a toxemic state with little or no dropsy. Of course, we expect dropsy in certain disorders of the blood and heart and in tubular rather than in interstitial renal inflammation or degeneration. Then, too, dropsy tends to withdraw soluble toxins from the circulation. But if dropsical effusions prevent toxemia, why are not the same poisons eliminated when the kidneys excrete freely? And why, without any definite line corresponding to the renal condition, do certain patients die of water-logging, without any marked nervous manifestations, while others, without more than a trace of œdema, have convulsions, and still others coma?

Renal Paresis is very frequent in disease of the digestive organs. Without any definite renal lesion the kidneys are more or less transiently deficient in function, even allowing for the diet in the particular case. In asthmatic and other patients, we may note an almost absolute paralysis of gastric and renal function for many hours. Food is vomited unchanged and the gastric filtrate has no acid, unless a little due to fermentation; there is no peptone ring and no curdling of milk or digestion of egg albumin. Meantime, no urine is formed. In Addison's disease, we may note gastric achylia, pancreatic and intestinal achylia, so far as may be demonstrated by our present rather crude methods, and the renal function may be much below par. In seasickness, there is a purely functional quiescence along the same lines.

Renal Supersecretion, commonly termed diabetes insipidus, is a matter that deserves careful scientific study. Apparently diabetes insipidus includes several conditions: (1) True diabetes in which, for some reason, sugar is temporarily absent. There may or may not be concomitant azoturia. Usually, the sugar and the nitrogen fall *pari passu.* (2) Unrecognized interstitial nephritis. (3) Cardiac and other complications causing increase of arterial pressure and consequent diuresis. (4) Excessive ingestion of salts and sugar, beer, etc., which lead to the absorption into the blood of unusual quantities of water; the sugar, especially, largely disappears, so that no more than the normal or a slightly exaggerated trace is found in the urine. (5) Excessive imbibition of water as such or in various beverages. (6) Central nervous or ganglionic disturbances which exalt the renal function.

It is obvious that the foregoing classification is inexact and that the different groups overlap. Only the last really deserves to be termed an essential polyuria or diabetes insipidus, and its explanation is at present far from satisfactory.

Perversions of the urinary secretion or, rather, excretion cannot be considered in detail. All things considered, the urinary examination is of more value to us than that of the stomach contents or, *a fortiori*, of the feces. The test for indican deserves special mention, as it is within the power of any practitioner, is sadly neglected, and is often the key to the whole situation.

HEART-BLOCK (ADAMS-STOKES DISEASE).

By Glentworth R. Butler, M.D.,

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The clinical syndrome known as Adams-Stokes disease has been recognized since its first description by Adams in 1827. It is only within recent years, however, that an increased knowledge of cardiac physiology and pathology has enabled an intelligent conception of its cause and a reasonably sufficing explanation of its symptoms. The subject has recently been discussed in detail by Ashton, Norris, and Lavenson.[1]

The facts and theories, to which they direct attention, serve to explain very exactly the manifestations occurring in the Adams-Stokes syndrome. The characteristic symptoms are: (1) Slow arterial pulse, 30, 20, or less per minute; (2) brief syncopal, apoplectiform, or epileptiform attacks; and (3) pulsation of the veins in the neck, these pulsations occurring at intervals more frequent than the radial pulse-beat, the ratio being from 2 to 1 to 4 to 1 or more. The parallelism between the phenomena resulting from experimental constriction of the auriculoventricular bundle and those occurring in Adams-Stokes disease is so close that it is quite safe to attribute the latter to an interruption of the normal communication from auricles to ventricles either by functional weakness or organic defect.

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[1] Amer. Jour. Med. Sci., 1907, cxxxiii, 28.

The case which I wish to report is that of a man, forty-three years of age. He was seen by me on two occasions through the courtesy of his physician, Dr. Joseph G. Leiter, of Brooklyn. The patient, a business man, had always been extremely temperate and regular in his mode of life, with no bad habits in eating or drinking. The family history presented nothing of interest. His personal history told of typhoid fever twenty years, and lobar pneumonia two years previous to his last illness. Since the typhoid fever, and perhaps before it, his pulse had always been slow. During his pneumonia, Dr. Leiter never found it over 42 per minute. His final illness, which began ten days before his death, was ushered in by fever (not higher than 101°), and pains in the larger joints. The temperature and joint pains rapidly subsided, but on the third, fourth, and fifth days he had a brief epileptiform convulsion, and the radial pulse went down to 28. Subsequently the convulsion seizures occurred more frequently until they became almost continuous in the last day or two; the pulse slowed down still more, varying from 14 to 20 per minute between the attacks. The seizures began with a marked pallor, the pulse dropping to 6 beats or less per minute, nearly twenty seconds elapsing between successive beats on one occasion. The eyes rolled up and to the right, the pupils dilated, the head turned to the right, the lips were cyanosed, and the arms became rigid and underwent slight clonic movements. The unconsciousness varied from partial to complete and was always brief. Just previous to the end of the seizure the pulse began to beat a little more rapidly and much more strongly, the face flushed, and consciousness returned. Vomiting occurred with some frequency before the attacks. Finally the patient passed quietly away.

During my two examinations, which were made during the last three days of the illness, the auricular pulsations in the veins of the neck were plainly visible. They varied from 80 to 130 per minute, and the auriculoventricular pulse-ratio was suggestive, not of a partial, but of a complete heart-block, the ventricular systoles bearing no definite relation to those of the auricles. In contradistinction to Stengel's experience, it was possible in this case to hear (with a Bowles stethoscope) faint fluttering sounds over the third interspace to the left of the sternum, which sounds were distinctly synchronous with the visible auricular pulsations.

The autopsy, made by Dr. Raymond Clark, assistant pathologist to the Seney Hospital, showed, in synopsis, the following findings: Hypertrophy and dilatation of the left auricle and ventricle and right auricle, and hypertrophy of the right ventricle; moderate mitral insufficiency; thickening at the base of the mitral cusps; and an increase in the amount of adipose tissue in the pericardium over the right ventricle at the junction of the auricles and ventricles and along the coronary arteries. Microscopically there was fatty infiltration and degeneration in the walls of the right ventricle, and, to a less extent, in those of the left ventricles; and no pathological changes in the coronary arteries. The kidneys showed an acute exudative nephritis with fatty changes; the liver a slight interstitial nephritis, with fatty degeneration of the liver cells, and a moderate venous congestion.

Careful sectioning of the bundle of His, compared with similar sections from a normal heart, showed that the bundle was infiltrated with fat, and had undergone fatty degeneration to such an extent that it was atrophied to not more than one-fifth of its normal size. The result of the autopsy in this case, therefore, tends to confirm the later theories in regard to the nature and causation of Adams-Stokes disease.

POSITION OF THE MOTOR AREAS OF THE HUMAN CORTEX.

By Hermon C. Gordinier, M.D.,

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Case I.—*A rapidly growing glioma of the right motor area (precental convolution) occupying the leg centre, the centres for the arm and face being destroyed by softening. Clinically focal Jacksonian attacks, usually beginning in the left leg, and not preceded by a sensory aura, the leg becoming totally, the arm and lower facial muscles partially, paralyzed as to motion but not as to sensation.*

Mrs. M., aged seventy-six years, was seen on August 10, 1900. Her father died of cancer of the stomach, her mother of pneumonia. She lost two brothers—one of pneumonia, the other of la grippe. Three sisters are living and well. There is no history of insanity or nervous diseases in the family.

The present illness began ten weeks ago with a spasm of the whole left side. This spasm began as a jerky motion of the leg, which was followed by similar movements of the arm and face. The spasm was preceded by no signal symptoms, such as numbness, tingling, or formication, nor by headache, nausea, or vertigo. These attacks, numbering from three to twenty daily, have been repeated ever since. They always begin in the leg or foot, extend upward to the thigh and then involve the arm and face. They never have been followed by loss of consciousness. Since the first unilateral convulsion, she has been slightly paralyzed on the whole left side. She never experiences numbness or pain in the extremities. She states that she is perfectly able to appreciate at all times the exact position of her limbs in space.

She is slightly built, weighs 110 pounds, and is 5 feet 4 inches in height. There is no œdema of the extremities or cyanosis. Physical examination shows moderate general arteriosclerosis and left ventricular hypertrophy. The urine is negative.

The ocular movements are normal. There is no constriction of the visual fields. The pupils are equal and react to light and accommodation. The optic disks are normal. The facial muscles appear symmetrical, but the left nasolabial fold is not so accentuated as is that of the opposite side. The tongue is protruded straight and presents no tremor or wasting. The special senses are normal. All movements of the left arm are somewhat limited and the arm is held in a position of slight contracture. The movements of the left lower extremity are very limited and the foot is held in an equinovarus position. The most careful tests fail to bring out the slightest evidence of ataxia. The tactile, temperature, pain, muscular, and stereognostic senses are normal. The patient is able to appreciate weights and recognizes immediately the position of her limbs in space. The left patellar tendon reflex and the Achilles tendon jerk are greatly exaggerated and there is a distinct ankle clonus on the left side. The Babinski phenomenon is marked. Because of the contracted condition of the muscles of the left arm, it is rather difficult to be sure of the deep reflexes; there is, however, a definite triceps tendon reflex present. The superficial reflexes show no particular alteration. The reflexes of the right side are normal. The visceral reflexes are perfectly controlled. Her mental condition is normal. Up to the present she has had no severe headache.

October 1, 1900. The patient's condition has remained unchanged, save for the development of severe headaches located chiefly over the right forehead and extending down the right side of the neck; they are occasionally accompanied by attacks of projectile vomiting. Complete motor paralysis of the left leg exists, together with paresis of the left arm and lower facial muscles of the left side. The tongue deviates toward the left. She still has an occasional Jacksonian attack, sometimes beginning in the leg and at other times in the arm, and involving subsequently the muscles of the face. The pupillary reflexes are normal, and the optic disks show no changes. Cerebration is somewhat slower than normal, but questions are answered correctly. The head is rotated sharply to the right side. The most painstaking examination fails to elicit the slightest sensory defect.

15*th*. The patient has lost control over the sphincters. She is very dull and lethargic. The headache is much more severe. The left arm is capable of slight motion. The leg is perfectly motionless. The deep reflexes are exaggerated and ankle clonus still persists. The head remains rotated sharply to the right and is held rigidly fixed. She has had no unilateral convulsions for a fortnight.

November 3. The patient gradually passed into a coma and died. A postmortem examination limited to the cranium was made this evening. The following are the notes: The vessels of the pia are injected, especially on the right side. The dura and pia are adherent over the upper part of the right Rolandic area, beneath which exists a hard, somewhat oblong, nodular mass, involving the upper part of the ascending frontal convolution, the bases of the first and second frontal gyri (Fig. 1), and the ventral part of the paracentral lobule (Fig. 2), which is also softened. The growth measures 3 cm. from above downward and 3.5 cm. transversely. The part involving the paracentral convolution is 1.5 cm. in its broadest diameter. The growth is absolutely limited to the upper part of the precentral gyrus, the part of the paracentral convolution corresponding to the median surface of the precentral gyrus, and the bases of the superior and middle frontal gyri. The part of the precentral convolution not occupied by the new-growth is softened, and there is an area of softening in the centrum ovale beneath the tumor mass, which extends forward into the prefrontal lobe. The ventricles appear normal. The arteries at the base are atheromatous. The anterior part of the dorsal limb of the internal capsule appears much grayer than the posterior part. Sections through the crura cerebri, pons, and medulla show no macroscopic changes. Sections of the growth show it to be a glioma.

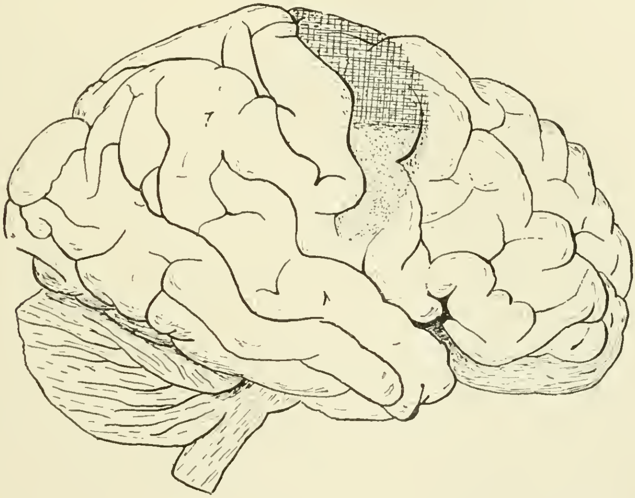

FIG. 1.—Schematic representation of the external surface of the right cerebral hemisphere (Case I). The heavy cross-lines indicate the position occupied by the growth, the dotted lines the area of softening.

The symptoms in this case clearly pointed to an intracranial lesion located in that part of the right motor area, the precentral gyrus, governing the movements of the leg, and subsequently involving the centres for the arm, face, and tongue. This focal diagnosis was supported by the complete absence of sensory symptoms and the presence of typical Jacksonian attacks, beginning in the leg and travelling to the arm and face, to be followed by partial and then by more or less permanent motor paralysis of the left leg, arm, and face, with contracture of the arm and leg, and marked increase of all the deep reflexes.

At first it was difficult to describe just what the pathological diagnosis was, as none of the general symptoms of a growth was present. Later, however, the onset and continuation of severe unilateral headache, together with occasional attacks of projectile vomiting without fever, despite the absence of optic neuritis, indicated that the lesion was probably a tumor.

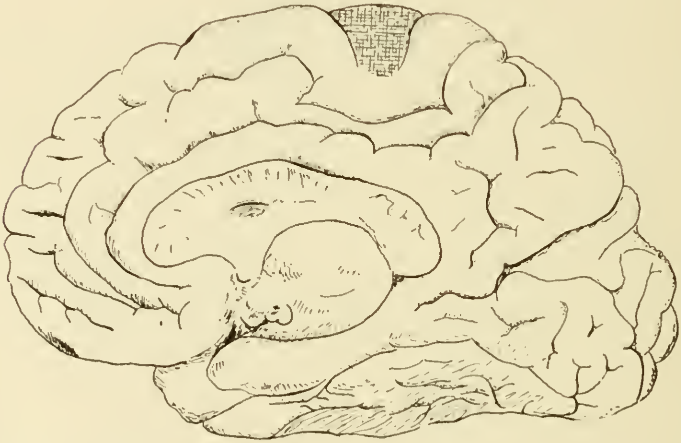

Fig. 2.—The position of the growth on the median surface of the right hemisphere (Case I).

Case II.—*An abscess of the right motor area (precentral convolution) characterized by unilateral convulsive movements of the muscles of the head and neck toward the left, together with similar movements of the arm and lower part of the face of the same side, without sensory auræ, followed by total paralysis of the left arm and paresis of the face.*

J. E. H., aged fifty-two years, married, a farmer by occupation, entered the Samaritan Hospital, February 17, 1900, under the care of Dr. E. D. Ferguson, who asked me to see him in reference to the possibility of an operation. His father died of rheumatism. His mother is living, but is rheumatic. One brother is living and well. No history of cancer, tuberculosis, insanity, or nervous disease in the family. The patient has had the usual diseases of childhood During the past two years he has had several attacks of indigestion. He denies venereal disease. The present illness began suddenly, January 5, 1900, with a severe unilateral convulsion followed by unconsciousness. The attack began with convulsive movements of the head and eyes toward the left side, then the whole left side of the body became convulsed and the patient lost consciousness. The effects of this attack passed away in a short time and no other occurred until January 31, 1900, the patient appearing perfectly well during the interim. Neither the first nor the second attack was preceded by numbness. The second attack began with convulsive movements of the head and neck toward the left, and almost simultaneously the arm and lower facial muscles of the same side were convulsed. This attack was not followed by loss of consciousness. A half hour later a third attack began—identical in character with the preceding one and without loss of consciousness. On February 14, the fourth convulsive paroxysm occurred, milder in character than the former, accompanied by movements of the muscles of the head, neck, arm, and face of the left side and without loss of consciousness. None of these paroxysms has been preceded by numbness or other sensory disturbances. After the last attack the patient noticed that the movements of his arm were very weak. A gradual failure of memory, particularly for names and dates, has accompanied these attacks. He has never experienced severe headaches, but complains of a constant dull frontal headache. He has noticed no loss of vision. He never vomits. The bowels are constipated; he urinates frequently at night, and has perfect control of the bladder and rectum.

The patient is moderately well built; height, 5 feet 10 inches; weight 160 pounds; healthy; musculature flabby; no cyanosis. Respiration, 16; pulse, 80, regular, full and of fair tension. The veins over the right side of the scalp are prominent. Movements of eyeballs and eyelids are normal. The pupils are midwide and react to light and accommodation. No contraction of the visual fields; vision is normal; the optic disks are normal; no defect of hearing. There is a left-sided, slight facial paralysis of central type, as evidenced by obliteration of the left nasolabial fold and dropping of the angle of the mouth on the same side; there is no rigidity of the neck; the tongue is protruded straight, and no atrophy or fibrillary twitchings were observed. The left arm is held in a position of semiflexion and is partially paralyzed. A slight contracture of the flexor muscles, which is easily overcome, is noticed. All movements of the left leg are normal. The triceps tendon reflex is present on the left side. No wrist tendon reflex or clonus on same side. The umbilical and epigastric reflexes are slightly exaggerated on the left side. The cremasteric is present, but not so active as on the right side. The left patellar reflex is exaggerated, and marked ankle clonus exists. The Babinski phenomenon is present. All reflexes are normal on the right side. The tactile, pain, and temperature senses are normal. The patient is able to recognize at once all objects handled. Cerebration is decidedly slower than normal. When a question is asked, some time elapses before the answer comes. He often errs in names and dates. No aphasic symptoms were present. The patient being right-handed no difficulty in writing was found. No unilateral convulsive seizures have occurred since the patient entered the hospital.

Physical examination of the internal organs is negative. The urine: Specific gravity, 1020; acid; no albumin, no sugar, no pus, no blood, no casts.

A diagnosis of tumor of the right motor area lying upon or just beneath the cortex was made.

February 22, 1900. The patient was anesthetized and the right Rolandic area was exposed after the method of Reid. No pulsation of the brain was noticed beneath the opening. The dura was incised and the vessels of the pia were found injected. No tumor was detected. The opening was then enlarged by a rongeur forceps more especially forward, but despite the large opening nothing was found save a circumscribed area which did not pulsate. This area was explored with fine needles, but meeting no particular resistance it was concluded that the lesion was subcortical and inoperable. The wound was then closed. No hernia cerebri followed. The following day a careful examination was made and the patient's condition was found to be the same as the day before the operation. The optic disks were normal. He did not complain of headache after the operation. A few days later it was noticed that the left arm was almost totally paralyzed. No sensory disturbances were present. No special change occurred in his general condition after the operation until March 12, when he became delirious, lapsed into an unconscious state, and died on the following day.

The postmortem was confined to an examination of the brain. The brain is normal in size and shape and the membranes are free save beneath the defect in the skull, where they are intimately blended, and the dura is adherent to the edges of the overlying (operative) bone defect. There is a slight increase of the cerebrospinal fluid. The right cerebral hemisphere presents a prominence in the region of the central convolutions especially in their upper half. Over this area exists a local collection of subarachnoidean fluid confined by delicate adhesions in the meshes of the pia-arachnoid. After removal of the pia-arachnoid there is found a tumor-like projection which involves the middle third of the anterior central or ascending frontal convolution, and extending forward and upward involves the bases of the superior and middle frontal gyri. This mass produced an evident bulging of the cerebral cortex, arising probably from the centrum semi-ovale, being covered only by a thin shell of cortical tissue half a centimeter in thickness. The mass has an elastic feel as if it contained fluid; it measures 4 cm. in its anteroposterior diameter and 3.5 cm. in its vertical diameter. The exact location of the mass (Figs. 3 and 4) is just beneath the cortex of the middle third of the ascending frontal gyrus, whence it extends forward and upward across the precentral sulcus involving the bases of

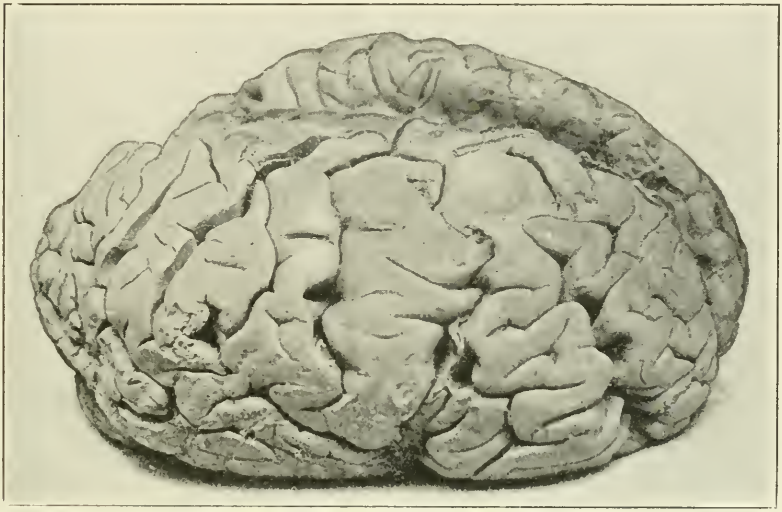

Fig. 3.—The external surface of the cerebral hemispheres, with a tumor-like mass involving the bases of the right first and second frontal convolutions together with the ascending frontal gyrus (Case II).

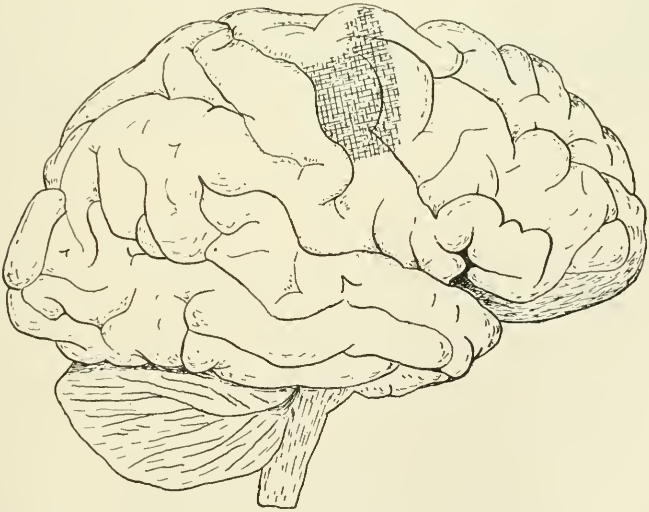

Fig. 4.—The exact position and extent of the abscess as it appeared on the external surface of the right cerebral hemisphere (Case II).

the first and second frontal convolutions, which gyri are pushed forward by it. The ascending frontal convolution is, from below, upward, distinctly widened. The cortical portion of the posterior central gyrus is not involved, but the fissure of Rolando in its upper part is narrowed. The superior portion of the ascending frontal, the paracentral lobule, the marginal portion of the superior frontal gyrus, as well as the outer and median surfaces of the superior parietal convolutions are normal.

After carefully hardening the brain in formalin, a series of frontal sections was made, whereupon an abscess was discovered in the right hemisphere, surrounded by a distinct capsule 0.5 cm. in thickness (Fig. 5). The abscess is entirely subcortical and measures, in its anteroposterior diameter, 5 cm.; in its vertical, 2.5 cm.; and in its broadest part, 3.25 cm. The abscess cavity extends forward into the frontal lobe involving the centrum ovale just beneath the cortex of the bases of the superior and middle frontal convolutions. It then broadens out beneath the middle of the anterior central convolution, where it has its greatest development. The superior wall of the capsule is covered by a thin shell of cortex, less than half a centimeter in thickness. The abscess proceeds backward, rapidly diminishing in size and depth, and terminates deep beneath the level of the middle third of the posterior central convolution.

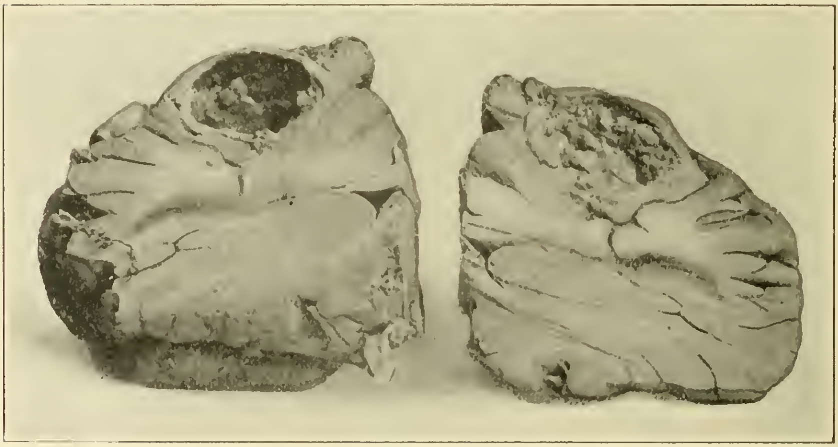

FIG. 5.—Frontal section through the frontal lobe at the precentral fissure and the middle of the ascending frontal gyrus (Case II).

The pus in the abscess cavity was thick, and contained much broken-down tissue. Coverslips taken from it showed a rod-shaped organism resembling one of the "colon" group, which completely decolorized with Gram's method. The identity of this organism could not be determined, as the brain was sectioned after several months of hardening in "formol" solution. The surrounding brain tissue showed only inflammatory changes, evidenced by the presence of numerous dilated vessels, with much small round-cell infiltration, especially in and about the perivascular spaces. The neuroglia elements were not proliferated.

Case III.—*Recurring clonic spasms of the facial muscles of the right side existing for three years, without aphasia, due to a lesion (scar) of the lower fourth of the left precentral gyrus.*

Mrs. E. F. R., aged fifty years, married. Has had five children, three of whom are living. Her father died at eighty-four years of age, of some obscure disease of the liver; her mother is living and is comparatively well. She suffers from rheumatism. Lost a brother in infancy by cholera infantum. Her husband died of phthisis pulmonalis. She has had all the usual diseases of childhood. She had always enjoyed good health until three years after marriage, when she was infected with syphilis by her husband. Two children were born after her infection, both showing unmistakable signs of congenital syphilis. One died shortly after birth and the other lived until the age of twenty-four years, dying then of tuberculosis. Her present illness began four years ago, when she became nervous and developed severe unilateral headaches, chiefly nocturnal in type, and confined to the left side. The headaches continued about three months, when there occurred a clonic spasm of the facial muscles of the right side. At first these attacks occurred only rarely, but they gradually increased in frequency, so that they recurred every day or two for a period of about three years. They were never preceded

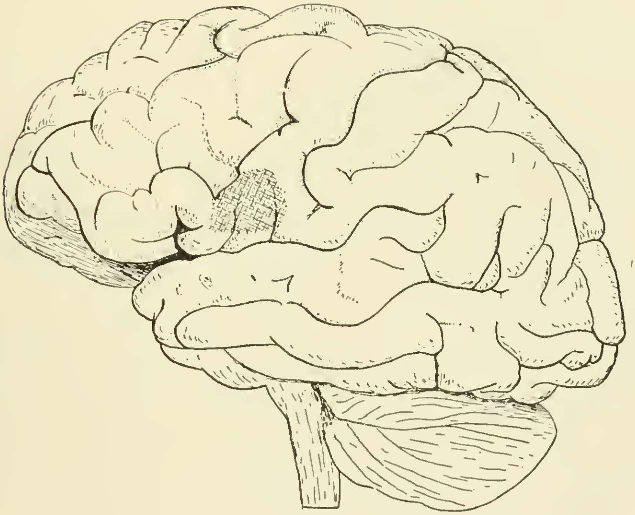

Fig 6.—The cross-lines indicate the position occupied by the lesion (syphilitic scar) in the left cerebral hemisphere, which excited recurring attacks of right facial monospasm (Case III).

by an aura nor followed by loss of consciousness. She never suffered from numbness, and stated that her vision was excellent.

Examination, October 1, 1900. She is rather short, moderately well built, and weighs 140 pounds. The lungs are negative. The cardiac dulness is increased to the left and the apex is displaced downward and outward. The gait is normal, and all movements of the extremities are perfectly performed. Sensation is unimpaired; no ataxia is present. The patient recognized at once the various objects placed into her hands. There is no mental defect and no aphasia or agraphia is present. The patellar tendon reflexes are lively, otherwise the superficial and deep reflexes showed no deviation from the normal.

The physician was advised to give her potassium iodide with mercurial inunctions. With this treatment there was a gradual improvement; the headaches became less severe, and the facial spasms, though never ceasing, became much less frequent.

I saw her again April 12, 1901, and found her suffering from severe headache. She was also very apathetic and drowsy, with marked failure of memory, but no aphasia. Her renal condition had advanced. The left ventricle is enlarged, the apex being located in the sixth interspace just to the left of the mammillary line. The radial and temporal arteries are thickened; the tension is increased; slight œdema exists over the tibiæ.

The urine: Light amber; specific gravity, 1.012; contains a trace of albumin and granular and hyaline casts.

A paresis of the lower facial muscles of the right side of the face exists. The tongue is protruded straight; there is no difficulty of deglutition. The muscles of the palate functionate normally and there is no loss of the sense of taste or smell. Hearing is perfect. The ocular movements are all preserved. The pupils are equal and react normally to light and accommodation. Vision is perfect. The optic disks are normal. There is no impairment of sensation on either side of the face. There is no tenderness on percussion of the scalp, and no rigidity of the neck.

November 3, 1901, she developed a series of general convulsions, epileptiform in type and died the same evening.

A postmortem, confined to an examination of the brain, was permitted. No change was found in the brain save a localized thickening of the meninges confined to the lowermost part of the left precentral gyrus, the cortex of which had been destroyed by a broken-down gumma and much scar tissue.

In these three cases, which presented during life focal convulsive movements, followed by partial or complete paralysis of motion, not preceded by auræ, and in which the most careful examination failed at any time to disclose the presence of sensory symptoms, the causes were lesions situated ventral to the fissure of Rolando, in the precentral convolution, together with the bases of the superior and middle frontal gyri.

Case I was a glioma destroying the upper third of the precentral gyrus and its paracentral annex, with the bases of the first and second frontal gyri, which at first excited crural spasm and monoplegia followed by paresis of the arm and face, the latter symptoms being probably due to subsequent softening of the remainder of the precentral gyrus.

Case II was an abscess of the middle of the right precentral gyrus and bases of the superior and middle frontal gyri, which caused unilateral convulsive movements of the head and face toward the left, together with similar movements of the left arm and face, followed by complete motor paralyses of the left arm and paresis of the lower facial muscles of the left side.

Case III. The lesion, a broken-down gumma with the formation of scar tissue and consequent atrophy of the cortex of the lower part of the left precentral gyrus, caused for a period of about three years right unilateral facial spasm followed by paresis of the face without motor aphasia; death probably was due to uremia.

In 22 cases, either with autopsies or in which the cortical area was exposed at operations, which I have collected from the literature, presenting during life motor paralysis, preceded or not by Jacksonian attacks and without sensory symptoms, the lesions were invariably located ventral to the fissure of Rolando, involving the precentral convolution together with, in several instances, the bases of one or more of the frontal convolutions.

In 3 of these cases a crural monoplegia was due to a lesion of the superior part of the precentral gyrus and base of the superior frontal convolution of the opposite side. In 6 cases irritative and paralytic symptoms, involving the muscles of the opposite side and unaccompanied by sensory disturbances, were due to circumscribed lesions of the middle portion of the precentral and extreme base of the midfrontal gyrus. In 3 of these cases the lesions were strictly limited to the middle of the precentral gyrus. In 7 of the cases focal convulsive movements of the muscles of the opposite side of the face, accompanied or not by facial paralysis, and without sensory symptoms, were due to limited lesions located in the lower part of the ascending frontal gyrus. In one of these cases motor aphasia was present, the lesion having encroached upon the base of the left third frontal gyrus. In another case pseudobulbar paralysis was present, and was due to lesions symmetrically placed in each hemisphere and limited to the inferior part of the ascending frontal and bases of the middle and inferior frontal gyri.

In 4 cases with partial or complete hemiplegia without sensory loss of "partial or complete hemianesthesia," the lesions were confined to the precentral convolution and paracentral lobule. In 1 case a gumma of the left first frontal convolution, bordering evenly upon the adjacent paracentral convolution, had caused hemiplegia without hemianesthesia.

In another case with frontal lobe symptoms, partial hemiplegia and tonic spasm of the right arm without sensory defect, two tumors were found to explain the symptoms: one at the base of the midfrontal lobule just ventral to the precentral fissure, the other in the ascending frontal gyrus limited in front by the precentral fissure and behind by the fissure of Rolando.

Dieulafoy has recorded a case of paralysis of the right arm and right lower facial muscles, with sensation unimpaired, due to hemorrhagic extravasation the size of a nut in the ascending frontal convolution of the left hemisphere dorsal to the third frontal gyrus. In addition to the above-mentioned cases, I introduce brief abstracts of the following cases taken from Ferrier's article on "Localization" in Allbutt's *System of Medicine*.

Lloyd and Deaver have reported a case of excision of a portion of the hand centre of the right hemisphere, which caused paralysis of the flexors of the left wrist and fingers and left half of the face. Three months after the operation, with the paralysis still existing, the patient was able to feel the slightest touch and was able accurately to recognize weights. He could not distinguish the form of objects because of the inability to move the fingers. Thermal sense was normal.

In a case of von Bergmann's of excision of a portion of the hand centre in the left hemisphere there was not the slightest affection of sensibility in any of its forms.

In Parker and Gotch's case of excision of a portion of the genu of the ascending frontal convolution of the right side, although followed by temporary paralysis of the left hand, no impairment of sensation resulted.

Diller and Buchanan have reported a case with paresis of the right limbs and right side of the face, due to subcortical cyst in the lower part of the ascending frontal convolution. The patient could feel and localize touch in any part of the paretic side and discriminate weights accurately.

Of great value in this connection in support of the doctrine of the definite localization of motor and sensory centres distinct from one another are the cases of partial or complete sensory loss without motor symptoms, due to lesions located dorsal to the fissure of Rolando in the parietal lobe. Redlich, from a study of the position of the lesions in 20 cases, with defects or loss of the muscle sense, believes that we can positively locate a centre for muscle sense in the parietal lobe. Von Monakow, Vetter, Grasset, and Starr have each recorded cases of great defect of the muscle sense without the slightest motor paralysis, with lesions confined to the parietal lobe. In 3 of these cases the central convolutions were found microscopically normal.

Interesting in this connection are the cases of von Monakow, Mills, Walton and Paul, Müller, Spiller, Russel, and Horsley, in which sensory defects of various combinations "tactile temperature, posture, astereognostic pressure, space," etc., unaccompanied by motor paralysis, were due to lesions in the parietal lobe.

It is also worthy of note that in 30 cases studied from the literature with definite motor and sensory paralysis due to cortical disease the lesions invariably involved both central convolutions.

In these days of experimental research this paper would be incomplete without alluding to the very interesting and valuable experiments of Tschermak and Professors Sherrington and Greenbaum, who, operating upon all known species of anthropoid apes, found that with unipolar faradization as the method of cortical excitation the responsive area was limited to the precentral convolution, and to the small coterminous portion of the paracentral lobule on the mesial surface of the hemisphere. Not once in the experience of Professors Sherrington and Greenbaum were movements obtained by excitation of the normal anthropoid postcentral gyrus, nor was experimental ablation of a portion of the same convolution attended by positive interference with motion.

These observations have been further supplemented and largely verified by the valuable experiments upon the cortex of the human brain exposed during operations by Keen, Mills and Frazier, Cushing, Lloyd, and others.

Furthermore, the very recent histological studies of Cajal and Campbell show that a marked difference of construction anatomically exists between the precentral and postcentral gyrus. And of great value in this connection are the studies of Campbell of the changes in the cerebral cortex in cases of amyotrophic lateral sclerosis and tabes dorsales. In the former, the type of a pure motor disease, he found definite changes limited to the precentral convolution, whereas in the latter, a purely sensory one, the pathological changes were limited to the postcentral gyrus.

From a careful study of my own cases and those that I have been able to collect from the literature I have formulated the following two conclusions:

1. There is a determined area in the cerebral cortex presiding over motor functions, and that area is the precentral convolution and its paracentral annex (possibly the bases of the superior and middle frontal gyri may have to be included).

2. The motor and sensory areas of the brain, though interdependent, are, as in the spinal cord, perfectly distinct from one another.

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A CONTRIBUTION TO THE PATHOLOGY OF REFRIGERATION FACIAL PALSY.

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In order that the pathogenesis of Bell's palsy may be definitely determined we must insist that the type of cases studied shall be uniform in clinical occurrence and symptoms. Bell's palsy *per se* is a refrigeration palsy occurring in individuals who have no other organic disease. This statement, however, must not rule out such chronic predisposing diseases as rheumatism. In the refrigeration Bell's palsy a rheumatic history may or may not be present; it is much oftener absent. In the Vanderbilt Clinic statistics it was ten times more frequently absent than present.

The cause of the great frequency of paralysis of the facial nerve, as in Bell's type of palsy, is still moot. It is, however, due probably to anatomical causes. The nerve has a tortuous and exposed course through the Fallopian canal. It is easy to understand that inflammation may readily extend from the middle ear, or that cold may operate through the same channel, causing a local neuritis and palsy. In further substantiation of the vulnerability of the facial nerve to exposure, we know the facial nerve is infrequently paralyzed when the face is properly protected by hair or clothing. Bell's palsy is very infrequently seen in animals. Furthermore, it is reasonable to suppose that a certain number of individuals suffer a congenital narrowing of the foramen or canal through which the nerve passes. It is known that the size of the foramen of the two sides differs in the same person and in different persons. Inasmuch as facial palsy occurs much more frequently in man than in animals, one is justified in assuming that the greater functional and structural development of the nerve in man is a factor in the frequent causation of the nerve palsy. It is also worthy of note in comparative anatomy that the soft tissues increase more rapidly in size than the bony parts in the process of evolution. This would explain why the facial nerve is often found very closely enveloped in its bony canal. Thus it is easy to account for the relative frequency with which the initial swelling of inflammation in the facial nerve (Fallopian neuritis) may induce secondary compression of the nerve strands in a rigid bony canal, and finally cause thereby a simple degeneration of the whole peripheral segment. This theory would explain the varying grades of completeness of palsy following a variation of the exciting cause.

No cases of refrigeration Bell's palsy have been studied pathologically to the time of this report. Before placing the results of a study of two such cases on record, a critical digest of the few cases which are often quoted to explain the pathology of Bell's palsy need to be disposed of: three cases figure extensively in literature—those of Minkowski,[1] Dejerine and Theohari,[2] and Alexander.[3] The findings in all are quite unreliable, as the examinations were made in all three some fourteen years ago, at a time when the distinction between the histological changes of inflammation and degeneration of peripheral nerves were not so definite as they are today. Indeed, many changes were then described as inflammatory which are known today to be only degenerative. Much confusion also resulted owing to imperfection of staining methods. The acceptance today of these earlier findings accounts in no small degree for the contradictory beliefs since held in regard to the initial lesions in all morbid processes of peripheral nerves.

The foregoing facts account in part for the hesitancy with which one accepts the purely degenerative changes noted in the Fallopian canal of Minkowski's case, as he expressly states that the cellular elements showed no changes that might be interpreted as inflammatory. He could hardly have overlooked the leukocytic and connective-tissue cell invasion seen in simple degeneration of divided nerves, which but a very few years ago were counted as inflammatory. These changes, although now known to be as common in inflammation as in degeneration, are nevertheless of a passive character. One must not only designate Minkowski's observations as inexact in analysis of the histological lesion, but also discredit the acceptance of the case at all, as his patient died from acute poisoning two months

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[1] Arch. f. Psychiatrie, 1891, xxiii.

[2] Cited by Minkowski from Compt.-rend. Soc. de biol. (reference omitted).

[3] Arch. f. Psychiatrie, 1901–2, xxxv, 783, and Arch. f. Ohrenheilk., 1904, lxii. 8.

after the supervention of palsy, a time at which distinct deposit of connective-tissue infiltration must have been unmistakably present. The faults in Minkowski's observations also prevent us from considering the case as in accord with the view of a degenerative neuritis or simple degeneration in the segment of the nerve anterior to the Fallopian canal, although he expressly defines a simple degeneration of the nerve in the periphery. The cellular changes were reported to be also progressively less as he proceeded centrally (evidence of non-regeneration). The degeneration was traced as far as the geniculate ganglion. The ganglion itself and the nerve above were, however, reported normal. We may, therefore, conclude definitely to exclude Minkowski's findings as a case of Bell's palsy with simple degeneration, as he does not appear to have given any attention to the state of the vascular perineural sheath, in which the most marked changes occur in inflamed nerves. The microscopic findings are certainly unique and unexplainable upon any basis for Bell's palsy as we know the lesion. One may also state in passing that the absence of nerve swelling in the canal may be explained on the ground that the examination was made more than two months after the initial neuritis, which is a sufficient time for the nerve swelling to have disappeared completely, had it once been present.

The facial palsy studied by Dejerine and Theohari occurred in a woman of eighty-one with uterine cancer—a complication which might easily vitiate its inclusion, in view of the fact that the palsy was preceded by a superficial cervical zoster, a condition now well known to be an infectious lesion of the posterior nerve ganglion. Further to complicate the clinical picture the woman died of an infectious pneumonia. While the condition of palsy of the facial nerve might in result be analogous to Bell's so-called refrigeration palsy, the lesion, as we shall note, must have been quite different. The findings in Dejerine and Theohari's case showed that the nerve was not compressed in the petrous bone, but that the entire facial nerve was the seat of parenchymatous neuritis, especially marked in the branches to the chin and the lower lip. A few degenerated fibers were found also in the chorda tympani. But few fibers of the nerve within the petrous bone were diseased, and the nerve roots were nearly intact. Many hyaline cells were found in the facial nucleus, and there was an absence of chromatin; the nucleus was nearly destroyed by disease. The case can hardly be considered in any light other than one of infectious neuritis of the whole peripheral system—nerve endings, trunk, root, and nucleus. The clinical and pathological state indicates conclusively that it was not one of refrigeration Bell's palsy, and the findings cannot explain the pathogenesis of Bell's palsy.

In comparing Alexander's case, on the one hand, with the cases of Minkowski or Dejerine and Theohari, we are struck with the essential difference in the anatomical changes in the two classes of cases. Alexander's occurred in a man aged fifty-six, who also suffered from cancer, but of the œsophagus. The intercurrent Bell's palsy underwent partial recovery in the four weeks preceding death by cancerous cachexia. A degenerative inflammation of the facial nerve and geniculate ganglion was found; the entire periphery was affected by degeneration. All the inflammatory changes were restricted to the nerve in the Fallopian canal and geniculate ganglion. The contiguous structures showed no change. Alexander makes no mention of examination of nerve roots or nucleus. Some doubt must be thrown upon the value of Alexander's case in interpreting the pathology of Bell's palsy, as the cause inducing the facial palsy probably was an infection from the cancer lesion (infectious facial neuritis).

Assuming Bell's palsy to be refrigeration palsy, one must discard all the nerve findings heretofore reported as such, as well as some six other cases studied in suppurative and necrotic mastoiditis attended or followed by seventh-nerve palsy. For the purpose of this paper a detailed analysis of the latter class is omitted, for obvious reasons.[4]

Probably few will deny that in the pure refrigeration palsy cases there is any evidence that cold ever paralyzes intramuscular nerve endings. It is certain, moreover, that paralysis of all the fibers in a nerve, and of no other fibers in the immediate neighborhood, is an index of nerve-trunk disease. There is no evidence on record that the trunk of the facial nerve is involved in inflammation after its emergence from the canal. While we tentatively accept the lesion of refrigeration facial palsy to be one of Fallopian neuritis, it must be admitted that this lesion is still unproved. This latter statement, however, does hold true for the condition of the trunk after emergence from the canal, as the two cases about to be reported as evidence testify. Further corroborative data must also soon be forthcoming as the surgical treatment of Bell's palsy by nerve anastomosis is more generally practised.

The summary of findings in two such cases of refrigeration palsy treated by faciohypoglossal anastomosis (operation by Dr. Taylor) is given as a preliminary contribution to the final disposition of the exact lesion in the peripheral trunk of the facial nerve of this type of palsy.

The palsy in the first case was typical refrigeration palsy of the right side. The paralysis was complete. There was no family or personal history of rheumatism or syphilitic taint. The woman in whom it occurred was forty-five years old. Reactions of degeneration remained complete, and *at the end of four months* faciohypoglossal nerve anastomosis was undertaken successfully. A small portion of the trunk of the facial nerve just below the stylomastoid foramen was preserved for microscopic study.[5]

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[4] The secondary neuritis of the facial nerve following mastoiditis, which has been so carefully described by May (Münch. med. Woch., 1884, No. 31), Darkschewitch and Tichonow (Neurol. Ctbl., 1893, x, 329), Flatau (ibid., 1896, xv, 718), and Meyer (Jour. Exper. Med., Nov., 1897), is quite different in onset, course, and pathology from that seen in Bell's refrigeration palsy. The lesion is much more severe and extensive. It is a parenchymatous (infective) neuritis, and its much graver prognosis is in marked contrast to Bell's palsy.

The second case of left facial palsy was also a typical refrigeration Bell's palsy. It occurred in a woman of forty-two, who was otherwise well. There was no rheumatic or syphilitic taint in the family or personal history. *In the twelve years following the onset* of the palsy no functional regeneration was noticed. On the other hand, there was complete atrophy of all the muscles supplied by the facial nerve above the angle of the mouth. Those at and below the angle of the mouth underwent considerable atrophy and contracture.

There was no spasm of muscle in these parts. Complete reactions of degeneration were obtained. The operation of faciohypoglossal anastomosis was undertaken by Dr. Taylor to correct the deformity of the lower part of the face, and to restore as much functional power as possible in these parts. The portion of the nerve trunk examined was taken in the same manner and from the same place as that of Case I.

A summary of the findings in the two cases is as follows: The two cases presented the condition found in a more or less acute and chronic state, respectively. In Case I (acute) the nerve trunk was in a condition of incomplete degeneration (Fig. 1). There were still a few nerve fibers undegenerated, or in which the myelin sheath was only partially broken up, and these stood out in marked contrast to the degenerated nerve fibers in which they were embedded, which latter show here and there fragments of myelin. There was, furthermore, a marked invasion of the tissues with leukocytes, and the cells of the neurilemma were shrunken and shrivelled in some portions; in others the nuclei of the neurilemma cells were large and were defined. The perineural sheath showed marked infiltration with leukocytes. The pathological picture shown was one of simple degeneration, with here and there slight evidences of a reparative process, like those shown most exquisitely in the recent exhaustive experimental studies by Ballance and Stewart.[6]

The picture in Case II (chronic) was in marked contrast to the preceding (Fig. 2). The nerve fibers were completely degenerated, showing but occasional granules belonging to the myelin sheath. There were no undegenerated fibers. The perineural sheath showed the most decided change. There was marked proliferation of the cells belonging to the perineural sheath, with infiltration of the tissues with clumps of cells scattered here and there throughout the section. This tissue exceeded the proper nervous tissue of the section by about two to one. The neurilemma cells were markedly shrunken and shrivelled. The picture, again, was one of complete degeneration, with no evidence of a repair process. If autogenous

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[5] The microscopic analysis of my two cases here reported was made by Dr. Thomas P. Prout, to whom my thanks are due for the report and the photomicrographs.

[6] The Healing of Nerves.

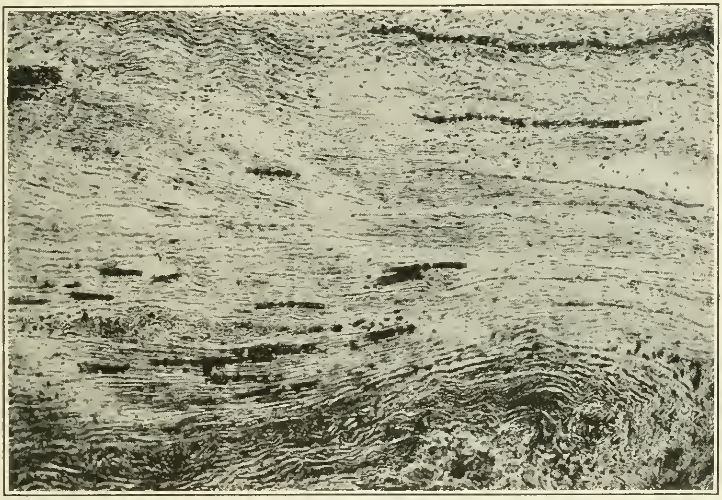

Fig. 1.—Case I, Mrs. B. G., aged forty-five years; acute refrigeration (Bell's palsy) of four months' duration. Photomicrograph of the facial nerve at the stylomastoid foramen, showing simple degeneration of the nerve fibers; here and there are seen neurilemma cells and a few regenerating nerve fibers, but no evidence of inflammatory lesion in the nerve. Peripheral degeneration following Fallopian (compression) neuritis.

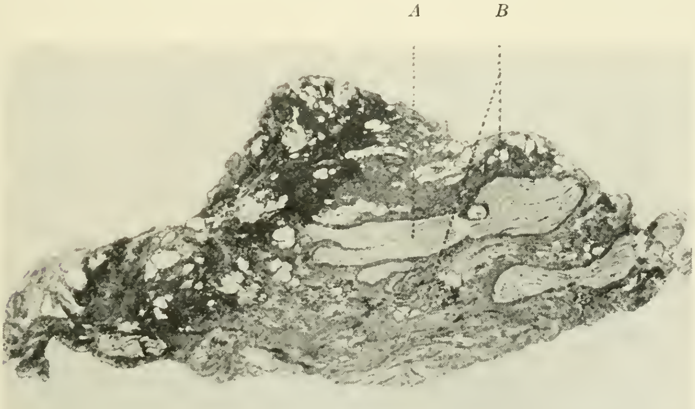

Fig. 2.—Case II, Mrs. M. J., aged forty-two years; chronic refrigeration (Bell's) palsy of twelve years' duration. No functional regeneration. Great atrophy and contracture of the muscles. Photomicrograph of the facial nerve at the stylomastoid foramen, showing simple degeneration of the nerve and connective tissue overgrowth. No attempt at repair. *A*, nerve fibers; *B*, connective tissues. No evidence of an inflammatory lesion. Simple peripheral degeneration following Fallopian (compression) neuritis.

peripheral regeneration (embryonic) had taken place in this case it must have disappeared, as no evidence of such was present in the specimen.

From a clinical viewpoint many data point to a simple degeneration of the peripheral portion of the facial nerve in Bell's palsy. The infrequency of pain[7] in the peripheral nerve distribution and the absence of tenderness upon pressure in either the nerve or the muscles indicate degeneration. Again, the passive movements of the muscles are not painful, as in neuritis. Certainly there are as few clinical as well as pathological reasons to indicate that the lesion in the peripheral portion of the nerve in Bell's palsy is one of parenchymatous neuritis. The clinical phases of recovery in refrigeration palsy of the facial nerve have many points in common with those seen in reunited or severely compressed peripheral nerves occurring elsewhere in the body.

Finally, I may summarize this study by saying: (1) Refrigeration Bell's palsy is in all likelihood a Fallopian neuritis; (2) the strands of the facial nerve are severely compressed by the initial inflammatory swelling in a rigid canal, and in this manner degeneration of the whole periphery of the nerve is induced; (3) the predisposition of some individuals to a Fallopian neuritis, and its consequent facial palsy, rests largely on anatomical grounds, which are mainly comprised in the fact that the nerve has a tortuous and exposed course through a bony canal, probably often congenitally insufficient in size.

THE CHANGES PRODUCED IN THE KIDNEYS BY RÖNTGEN IRRADIATION.

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The publication during the last two years of Heineke's important work upon the changes produced in the lymphoid tissues by Röntgen irradiation and the observations of Albers-Schönberg upon the disturbances of testicular function similarly produced, have effectually disposed of the dictum that had been rather vigorously upheld that Röntgen-rays produce changes only in the superficial parts of the body exposed to their action. While other investigators have confirmed the observations of these writers and have added to our knowledge concerning the changes in the lymphoid tissues and sexual glands, we are still practically ignorant of the effects of Röntgen irradiation upon the other internal organs. The few investigations made along these lines have been rather of the nature of casual or incidental observations, and do not lead us to any definite conclusions. Particularly with reference to renal changes due to Röntgen irradiation, are we still in the dark, and no thorough investigations have been carried out in this field. In fact, but two observations relating to this subject are to be found in the literature, those of Linser and Baermann and Buschke and Schmidt.

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[7] Pain when present is usually confined to the ear or its immediate neighborhood (probably due to the compression neuritis in the bony canal).

Linser and Baermann[1] found that exposures of one hour produced transitory albuminuria without apparent changes in the renal epithelium. Buschke and Schmidt[2] could discover no disturbances of function or pathological changes in the kidneys of guinea-pigs and rabbits exposed percutaneously for periods varying from five minutes to two hours, although some of the animals died without any apparent cause during the first week after the exposure. In animals in which the kidneys were laid bare by incision and freed from their fatty capsules, irradiation with a medium tube for from one-half to two hours produced in some cases a slight albuminuria during the first week. In two cases only were definite histological changes found, the animals dying without apparent cause forty-five and fifty-six days respectively after the irradiation. In the kidneys of these animals extensive necrosis of the cortical substance was found, the necrosis extending even into the medullary pyramids. The neighboring tissues showed a marked reactive inflammation. Other animals similarly exposed lived six to eight weeks, showing when killed neither macroscopic nor microscopic changes in the irradiated kidney.

From these somewhat unsatisfactory and inconclusive experiments Buschke and Schmidt conclude that the kidney epithelium is much more resistant to the action of Röntgen rays than is the epithelium of the testicles. Based upon this comparison they advance the view that cells capable of speedy regeneration are less resistant to the rays, while those more slowly regenerating offer a greater resistance. Since the experiments of Linser and Baermann were practically negative in so far as the production of histological changes in the kidneys was concerned, and as only two of the animals used by Buschke and Schmidt showed pathological changes (and these changes might well be the result of the operative manipulations), it will be seen that at the present time there is practically no conclusive proof that renal changes can be produced by Röntgen irradiation. Clinical observations have been made of the occurrence of nephritis after irradiation, but the anatomical changes are unknown.

I was led to the present investigation by the remarkable kidney changes discovered in the material from two cases of leukemia that had been treated for some time with Röntgen rays. The first of

[1] Fortschritte aus dem Gebiete der Röntgenstrahlen, December 9, 1904.

[2] Deut. med. Woch., 1905, xxi, 495.

these cases was a patient of Drs. Brown and Jack, of Decatur, Illinois. The leukemia was of the myeloid type, the white cells numbering 800,000, and the spleen being greatly enlarged. Röntgen irradiation of the splenic region twice a week for two months produced a great improvement, the spleen becoming reduced in size and the number of white cells diminished. Daily exposures were then made for seven months, and the improvement was so marked that hopes of a complete cure were entertained. The patient remained in relatively good condition for six months although the leukocyte count was high and there was a high percentage of myelocytes. Röntgen irradiation was renewed when there was a change for the worse. The treatment was then continued for seven weeks, but the man gradually grew worse and died with symptoms of a severe intoxication. The autopsy was performed by Dr. Jack, and the material sent to me for examination. It had been fixed in 4 per cent. formaldehyde solution, and was embedded in paraffin and stained with various stains.

Microscopic examination of sections of the lungs, liver, pancreas, and large and small intestine showed atrophy, chronic passive congestion, and slight parenchymatous degeneration, but no signs of a leukemic condition. The white cells in the bloodvessels were not increased and there were no leukemic infiltrations. The spleen did not present the characteristics of a leukemic organ. Only in the retroperitoneal lymph nodes were changes found suggestive of an intrinsic disease of the blood-cell forming organs. These nodes showed a marked lymphoid hyperplasia, the normal structure of a lymph node being wholly lost. The lymphoid cells infiltrated the capsule and the surrounding adipose tissue. In so far as the pathological picture was concerned the leukemic condition had been changed into an aleukemic condition.

Without considering the other pathological features of this case, certain remarkable changes in the kidneys demand our attention at the present time. On cutting the kidney into pieces suitable for embedding, marked grating and resistance were noted. The cut surface showed numerous whitish areas and stripes corresponding chiefly to the medullary rays in the cortex and the collecting tubules in the medullary pyramids. The sections were cut with great difficulty owing to the resistance offered by these whitish areas. When stained the sections showed the presence of extensive deposits of lime-salts filling up the tubules and replacing the renal epithelium (Figs. 1 and 2). The deposits stained deep violet with hematoxylin, and dissolved slowly in 1 per cent. hydrochloric acid without the formation of bubbles. Although found chiefly in the distal convoluted tubules and in the straight tubules, the deposit was present also in many of the proximal portions of the convoluted tubules and glomeruli. The remaining portions of the parenchyma were atrophic and presented more or less marked cloudy swelling and fatty degeneration.

The intertubular connective tissue was increased and there were localized inflammatory changes throughout the cortex. The blood-vessels showed a condition of chronic passive congestion. The extent of the calcification was most remarkable, the greater part of the renal structure being replaced by the deposits. The condition resembled that sometimes seen after poisoning with mercuric chloride.

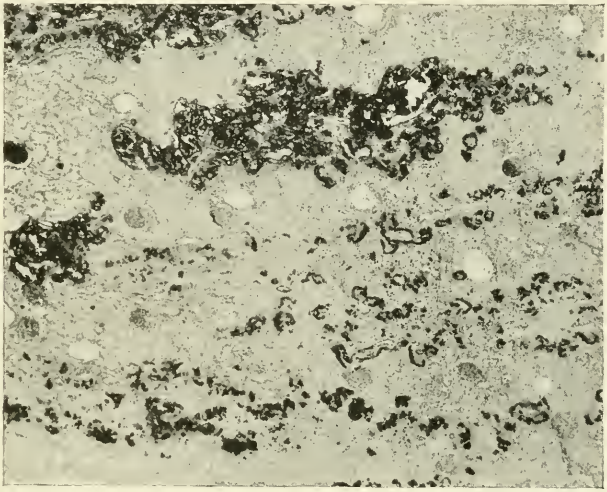

Fig. 1.—Deposit of lime-salts in the kidney from a case of myeloid leukemia treated by Röntgen irradiation. Hematoxylin-eosin preparation. The dark areas represent masses of lime-salts stained deep violet blue with the hematoxylin.

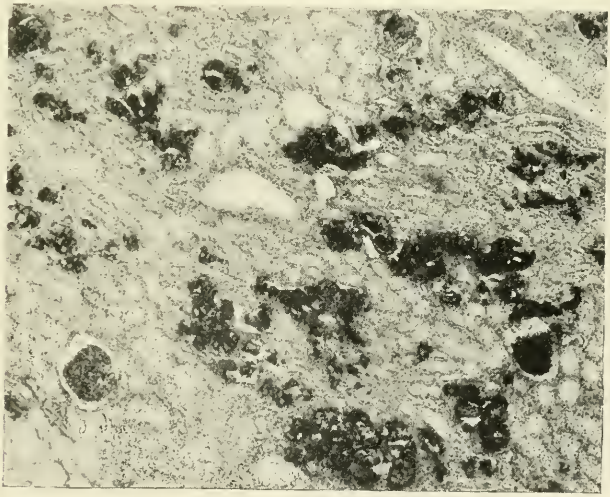

Fig. 2.—Higher magnification of Fig. 1. The deposit of lime is chiefly in the straight tubules and distal portions of the convoluted tubules, although all parts of the tubules, including the glomeruli, are involved. Hematoxylin-eosin preparation.

When dissolved out the lime-salts left behind a granular albuminous detritus, the renal epithelium of the affected tubules being completely necrosed.

In a second case of leukemia treated by Röntgen rays similar deposits of lime-salts were found in the kidneys. The patient, from Dr. Dock's clinic, University Hospital, presented universal glandular enlargement with a blood picture of a small-cell lymphatic leukemia. Thirty treatments in all of ten-minute exposures with a medium tube, six to eight inches distance, were given every other day to the cervical and axillary regions. The inguinal regions received nineteen treatments. The enlarged glands became much reduced in size, and there was at first a decrease in the number of leukocytes, followed later by a rise. While the general condition seemed at first somewhat improved, the patient soon grew worse and died rather suddenly with symptoms of intoxication. The autopsy was performed by me. Considering here only the kidneys, they were enlarged, pale, and soft. On section the straight tubules of the medullary pyramids appeared as whitish streaks and spots that could be scraped away as a finely granular chalky substance having a distinctly gritty feel. On microscopic examination the renal epithelium showed slight degenerative changes and there was local inflammatory reaction. Cloudy swelling and fatty degeneration were most marked in the distal portions of the convoluted tubules and in the straight tubules, the lumina of many of these being filled with casts of a finely granular calcium phosphate that, dissolved out with acid, left a granular albuminous cast. In all respects the condition resembled that seen in the first case, but much less advanced.

The extensive renal degeneration and calcification found in these two cases of leukemia, treated by Röntgen rays, excited at once interest in the question as to the possibility of some relation between these changes and the treatment. That such changes may be found in cases of leukemia not treated by irradiation does not appear in the literature. I have also examined the kidneys from twenty-five cases of leukemia (myeloid, small and large-cell lymphatic, chloroma, and the leukemic forms of lymphocytoma). In all these more or less extensive parenchymatous degeneration was found in the kidneys, as well as leukemic metastases, localized inflammatory changes, etc., but in none was there any deposit of lime-salts seen or such extensive necrosis. In general it may be said that the amount of parenchymatous degeneration found in the kidneys of leukemic cases is in direct proportion to the number of degenerating white cells seen in the blood, spleen, bone-marrow, and lymph nodes of a given case. The most extensive renal changes were found in two cases characterized by marked leukolysis.

The relationship apparently existing between the renal changes and leukocyte destruction, as shown by a study of these twenty-five cases, may be taken as an evidence that the more severe renal changes in the two cases of irradiated leukemia are the results of some poison arising from the excessive destruction of white cells by the x-rays. This view is supported by the pathological findings in a third case of leukemia treated by Röntgen rays. A patient of Drs. Grosh and Stone, of Toledo, Ohio, had myeloid leukemia for two years, the white cells being 226,500, and the spleen showing the usual enlargement. He was given five-minute exposures with a medium tube at a distance of six to eight inches for six weeks. The white cells were reduced to 10,600; the myelocytes from 52 per cent. to 2 per cent., and the spleen became smaller. The patient then died suddenly with signs of a severe intoxication. The various organs presented the picture of a myeloid leukemia with excessive leukolysis. The spleen in particular showed great numbers of disintegrating white cells and numerous areas of complete necrosis. The renal epithelium presented the appearances of advanced cloudy swelling, fatty degeneration, and simple necrosis. The bloodvessels were much congested. No deposits of lime-salts were present. The collecting tubules contained numerous hyaline and granular casts.

As it seemed very probable that the extensive kidney lesions in these three cases of irradiated leukemia bore some definite relation to the treatment and the resulting destruction of white cells, it was determined to carry out some experimental work bearing upon this point. The results are given below.

EXPERIMENTAL INVESTIGATIONS. *Technique.* The animals used were white mice and rats, guinea-pigs, rabbits, and Belgian hares. Percutaneous exposures were given, since all other sources of damage to the kidneys were to be eliminated, and it was also desirable to compare the percutaneous effects of the rays upon the kidneys with those upon the lymphoid tissues. The small animals were placed wholly in the field of exposure, while in the larger ones the field of irradiation was limited to the abdominal region including the spleen and kidneys. A medium soft tube was used, distance six to eight inches, four-inch spark gap, current about 3 milliampères, penetration power equal to 4 millimeters of aluminum. The animals were either killed by being opened under the influence of chloroform or were allowed to die spontaneously. In both cases the organs were removed from the body while warm and fixed at once in a saturated solution of mercuric chloride or in Flemming's solution. In a few cases in which the animal was not seen at death, the organs were fixed in a 4 per cent. solution of formaldehyde. With the exception of that fixed in Flemming's solution, all the tissues were embedded in paraffin and stained with various stains, hematoxylin and eosin being chiefly used.

Experiment 1.—White mouse exposed one-half hour. No apparent effects from the exposure. Killed at once. Autopsy appearances negative. The microscopic examination showed nuclear fragmentation of lymphocytes in the spleen. Very slight changes were found in the nuclei of the renal epithelium, consisting of a slight swelling and vacuolation of the nucleus, the chromatin particles being somewhat more compactly clumped. On the whole these changes were so slight as easily to escape notice, and would not ordinarily be regarded as pathological or as due to the exposure. Only when viewed in the light of careful control comparisons was the conclusion reached that they were the result of the irradiation.

Experiment 2.—White mouse exposed as above and killed one-half hour after the exposure. The gross appearance of the kidneys was negative. The microscopic examination showed some slight changes in the renal epithelium. The spleen contained a greater number of disintegrating lymphocytes.

Experiment 3.—White mouse exposed as above. No apparent effects. Killed one hour after the exposure. No macroscopic changes in the kidneys. The microscopic examination showed numerous disintegrating lymphoid cells in the spleen. The same slight changes were found in the renal epithelium.

Experiment 4.—White mouse exposed as above, and killed an hour and a half after the exposure. No symptoms. The macroscopic appearances of the kidneys were negative. The spleen was enlarged and dark red. The microscopic examination of the spleen showed great numbers of chromatin fragments throughout the pulp and follicles. The same slight changes in the chromatin of the renal epithelium were found but appeared less marked.

Experiment 5.—White mouse exposed as above and killed two hours after exposure. No symptoms. Macroscopic appearances of kidneys negative. Marked nuclear disintegration in the spleen. Microscopic changes in the kidney same as in the preceding.

Experiment 6.—White mouse exposed as above and killed two and a half hours after exposure. No symptoms. Macroscopic and microscopic conditions in spleen and kidneys same as in the preceding.

Experiment 7.—White mouse exposed as above. No symptoms. Killed three hours and twenty minutes after exposure. Spleen red; slightly enlarged. No macroscopic changes in kidneys. The nuclei of the renal epithelium showed such slight changes as to be practically normal.

Experiment 8.—White mouse exposed as above. No symptoms. Killed four hours after. Spleen slightly enlarged. No macroscopic changes in kidneys. Microscopic picture same as in preceding, with slightly more disintegration in the spleen.

Experiment 9.—White mouse exposed as above. Four hours after exposure the animal was more quiet than normal and its eyesight was apparently impaired. Back humped. It was killed five hours after exposure. The spleen was somewhat swollen and brownish in color. No macroscopic changes in the kidneys. Microscopic examination showed slight cloudy swelling of the renal epithelium.

Experiment 10.—White mouse exposed as above. Several hours after exposure became quiet, back humped, and eyesight impaired. Was killed six hours after the exposure. The macroscopic and microscopic pictures were the same as in the preceding.

Experiment 11.—White mouse exposed as above. Several hours after the exposure developed the same symptoms as in the preceding. Was killed seven hours after exposure. The spleen was small and more brown than normal. The microscopic examination showed slight cloudy swelling of the kidney epithelium. Numerous phagocytes containing chromatin granules were scattered through the spleen pulp, and the number of lymphocytes was diminished.

Experiment 12.—White mouse exposed as above. Same symptoms. Killed eight hours after the exposure. Macroscopic and microscopic pictures the same as in the preceding.

Experiment 13.—White mouse exposed as above. Same symptoms developed a few hours after exposure. Killed fifteen hours after the exposure. Macroscopic appearances negative. Slight cloudy swelling in renal epithelium. The greater part of the disintegrated lymphoid cells in the splenic pulp and follicles were collected in large phagocytes.

Experiment 14.—White mouse exposed as above. Same symptoms developed. Was killed twenty-four hours after the exposure. Macroscopic appearances negative. Slight cloudy swelling of renal epithelium, less marked than in preceding. Fewer evidences of lymphoid disintegration. Beginning regeneration.

Experiment 15.—Young white mouse exposed for five hours. Almost immediately developed symptoms, shivering, blindness, body damp and cold, back humped, and hindquarters paretic. Became comatose and died spontaneously twelve hours after the exposure. Body very dry, shrunken, blood thick, coagulating immediately. Spleen very small and deep brown. Kidneys deep brown, small and dry. The microscopic examination showed great destruction of lymphoid cells in the spleen, lymph nodes, and bone-marrow. The kidney tubules were distended, the epithelial cells smaller than normal, but staining more deeply, their nuclei pale and vacuolated. Heavy precipitate of albumin in the tubules.

Experiment 16.—Half-grown white mouse exposed for five hours. At the end of the exposure the animal appeared very ill, back humped, eyes closed, body damp and cold, diarrhœa, paresis of hindquarters, etc. Died in twenty-four hours of slowly progressive weakness and coma. Spleen small and dark-red. Kidneys small and deep-brown. All tissues very dry. Microscopic examination showed marked destruction of lymphoid tissues. The nuclei of the renal epithelium pale and vacuolated, the cell-protoplasm diminished and granular, the tubules distended and filled with a heavy albuminous precipitate.

Experiment 17.—Young gray mouse. Exposed five hours. Same symptoms as in preceding experiment. Died of paresis and coma forty hours after exposure. Macroscopic and microscopic findings same as in the preceding.

Experiment 18.—Full-grown white mouse. Exposed five hours. Developed the same symptoms more slowly and died on the third day. Spleen very small and dark red. Kidneys small and very brown. Microscopic examination showed marked destruction of the lymphoid tissues. The renal epithelium was smaller and stained more deeply than normal and its nuclei showed vacuolation and clumping of the chromatin. The tubules were distended and filled with a granular precipitate of albumin.

Experiment 19.—Full-grown white mouse exposed for thirty-two hours and fifty-five minutes during a period of five days. Died while being exposed, developing the same symptoms as the preceding. The autopsy showed great distention of the intestines, the peritoneum was very dry, the liver small and brown, and the spleen about one-third normal size, and very firm and brown. The kidneys were diminished in size and very brown. The microscopic examination showed the renal epithelium to be smaller than normal, very granular and staining heavily with eosin. The nuclei were pale and vacuolated. The tubules were distended and filled with a heavy granular precipitate. The changes in the kidneys of this case were much more severe than in any of the preceding, and were easily recognized.

Experiment 20.—Two white rats, one full-grown white mouse, three half-grown white mice, and one young gray mouse were exposed in the same field for five hours. A medium tube was used, backed, 6 ampères through the primary of coil, four-inch parallel spark gap, about 2 milliampères through the tube; penetration equal to 16 millimeters of aluminum. All the animals showed marked symptoms within twelve hours after exposure, similar to those described above. The large white rats seemed more affected than the full-grown mouse. The young white mice died within thirty hours; the gray one forty hours after exposure. The white rats died by the eighth day; the white mouse lived until the tenth day. The autopsy appearances were negative, or the same slight changes mentioned above. In the animals dying soon after the exposure the slight nuclear changes were alone found in the kidneys; in the animals dying later the renal epithelium showed a more marked condition of cloudy swelling, the degree of the kidney lesion corresponding in general to the severity of the symptoms and the length of time between the exposure and death, the renal changes being most marked in the animals dying last. Marked destruction of the lymphoid tissues was present in all the animals, but was more extensive in the white rats.

Experiment 21.—Twelve young but full-grown rats were exposed in the same field for five hours under conditions identical with those of the last experiment. One animal was killed daily for six days; the others were allowed to die, death occurring in all cases by the eleventh day after exposure. The symptoms in all were identical with those described above, gradually increasing emaciation, paresis, and coma. The lymphoid tissues showed extensive disintegration of the lymphoid cells during the first five or six days, the chromatin granules being gathered up by phagocytes and gradually removed after this time. Immediately following the exposure very slight nuclear changes in the renal epithelium were found; in the animals dying later the kidneys were enlarged and congested. The microscopic examination showed a marked cloudy swelling.

Experiments 22 to 40.—A series of experiments was also carried out with larger animals, rabbits, etc. It is not thought necessary to give the details of these experiments, since they do not differ from those above. The results were similar. Exposures of one-half to one hour produced very minute changes in the renal epithelium apparently quickly recovering. Prolonged exposures causing symptoms (it was not possible to give exposures sufficiently prolonged to cause the death of the larger animals) were followed by a condition of cloudy swelling of the renal epithelium and an albuminuria in proportion to the degree of destruction of the lymphoid tissue. Two exceptions to this were noted in two Belgian hares, exposures of one-half and one hour respectively causing immediate albuminuria and cloudy swelling.

Conclusions.—1. From the above it will be seen that percutaneous Röntgen ray exposures of small animals for half an hour to an hour produce at once slight nuclear changes in the renal epithelium, characterized by swelling of the nucleus, vacuolation, clumping of the chromatin, and some loss of staining power. Recovery from this primary injury to the chromatin is prompt, but there follows, after six to twelve hours, a secondary kidney change characterized by albuminuria and cloudy swelling. This second change occurs during the period of phagocytosis and removal of the lymphoid detritus from the spleen, bone-marrow, and lymph nodes, and appears to be in direct proportion to the degree of lymphoid destruction. It is, of course, possible that this second kidney change is but the result of the primary disturbance of the nucleus, but the first explanation seems to me the more probable.

2. In the case of an animal exposed continuously until death occurs the renal changes are more marked, the renal cells being diminished in size, cloudy, and the tubules distended with an albuminous precipitate.

3. Continuous exposures of five hours or more are invariably fatal to small animals, death occurring within ten days following marked symptoms of paresis and coma. These symptoms also bear a definite relation to the amount of lymphoid destruction and to the kidney lesion. To what extent they are dependent

upon direct injury to the central nervous system cannot be said at the present time. The character of the symptoms suggests a primary or secondary damage to the central nervous system. The assumption of a primary injury is upheld by the fact that all small animals exposed for five hours or more became either totally or partially blind. On the other hand, the progressive development of the symptoms some days after the injury is very suggestive of an intoxication, and the character of the symptoms accords with that of poisonings due to products of protein destruction.

4. If we may make practical application of these conclusions it would seem that the destruction of great numbers of leukocytes, as occurs in the Röntgen-ray treatment of leukemia, may be dangerous to the organism, either in its action upon the central nervous system or upon the kidneys. As to the occasional irradiation of the human body it is hardly likely that any serious damage to the kidneys could result, but in the case of repeated and prolonged irradiation of the lymph nodes and spleen the possibility of renal injury should be borne in mind. Careful and repeated examinations of the urine, therefore, should be made in all such cases. In the case of preëxisting nephritis a dangerous exacerbation may be excited by irradiation.

5. It is probable that the Röntgen rays cause disturbance of the chromatin of all cells, the lymphoid cells and the epithelial cells of the testis being the most susceptible. As compared with these the renal cells are much more resistant. In a general way, then, the view is upheld that all cells capable of rapid proliferation or renewal are especially susceptible to the action of Röntgen rays.

Note.—Since this paper was presented before the American Röntgen-ray Society, in August, 1906, I have examined the autopsy material from a case of Hodgkin's disease treated for some time with Röntgen irradiation and have also performed an autopsy upon a case of myelogenous leukemia similarly treated. In both, but particularly in the leukemic case, the kidneys showed the same condition of "chalk infarct" and calcification described above. There can be little doubt, then, that this condition is the direct result of the treatment. Its greater extent in the two cases of myelogenous leukemia is apparently in proportion to the greater destruction of white cells. Krause and Ziegler[3] recently have practically repeated the experiments herewith detailed. These investigators were unable to discover any changes in the kidneys, after short or prolonged irradiation in mice, rabbits, guinea-pigs, rats, and dogs, that they could positively ascribe to the action of Röntgen rays. In conclusion, it may be repeated that the very minute changes caused by irradiation in the renal cells may be wholly missed unless the fixation of the tissue is perfect and careful comparison with normal renal epithelium similarly fixed is made.

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[3] Fortschritte aus dem Gebiete der Röntgenstrahlen, 1906, x.

THE PATHOGENESIS OF REFLEXES, APROPOS OF A CASE OF TUBERCULOMA OF THE SPINAL MENINGES.

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The case which I report herewith presents some unusual features and suggests some inferences in regard to the pathogenesis of reflexes:

A. F., a colored girl, aged eighteen years, was admitted to the Philadelphia General Hospital, October 18, 1905, with the following history: About six months prior to her admission she noticed that her right arm was becoming weak and painful. Two weeks later the same condition developed in the left arm and shoulder. A month later, weakness without pain appeared in the lower extremities; this weakness gradually increased, so that within a month there was complete paralysis of the legs and of the right arm. At the same time there was loss of control of the bladder and rectum.

Upon examination there was found some preservation of power in the left arm, but the other three extremities were totally paralyzed. Wasting of the entire musculature of the body and especially of the extremities was quite pronounced. The paralysis of the legs was flaccid. The knee-jerks and Achilles-jerk were abolished; there was no ankle clonus, no Babinski sign. Test for sensations revealed that pain and touch sense were abolished in both legs as far up as the hips; the rest of the body presented normal sensations. (This statement is not absolute, as her answers varied from time to time.) There were multiple bed-sores. The pupils were unequal, the left being the larger; they reacted sluggishly to light and accommodation. Percussion and auscultation gave typical signs of tuberculous lungs. At no time did the patient complain of pain. She died two days after admission.

The postmortem record shows tuberculosis of the pleuræ and lungs, brown atrophy of the heart, and chronic interstitial nephritis. On the right side of the seventh cervical vertebræ was found a yellowish-gray mass. When the spinal canal was opened, a large cheesy-like mass was seen extending from the fifth cervical to the second dorsal vertebra on the right anterolateral surface of the dura. The dura with the growth was adherent to the bone. The vertebræ were softened; the greatest destruction was between the first dorsal and seventh cervical. On opening the dura the latter was found greatly thickened, its inner surface was smooth and glistening; the pia-arachnoid was smooth; the subarachnoid space contained a small amount of clouded yellowish fluid.

Microscopic examination shows that the greatest amount of damage done to the cord was at the lower cervical and upper dorsal region. (Fig. 1). On the side of the tumor is seen not only degeneration but also total destruction of the largest part of the white substance, particularly at the periphery, so that the nervous tissue is completely separated from the adjacent pia-arachnoid. There are a great many vacuoles and clefts in this portion of the white matter. The axis-cylinders in this area are either swollen or absent. The vacuoles are the result of complete destruction of the nerve fibers. On the opposite side the degeneration is confined to the entire periphery of the cord. Light, softened areas are also found in the posterior columns and in the white matter between and in front of the anterior cornua. The cornua themselves are deformed, very much diminished in size, with marked degenerative changes in their cells. Above this level, that is, in the upper cervical segments, there are distinct degenerated areas confined to the posterior colums, direct cerebellar, and partly Gowers' tracts (Fig. 2). This ascending degeneration extends upward as far as the first cervical segment, but no trace of pathological changes can be found in the medulla, in which the fillet and the restiform bodies are normal. The lower dorsal and lumbar segments of the cord show a descending degeneration in the crossed pyramid tract.

The most interesting changes are those of the meninges, particularly of the dura. Microscopically, the tumor was extradural and originated in the vertebræ. At the level of its maximum development the microscopic examination shows a very pronounced tuberculous pachymeningitis, invading not only the external aspect of the dura, but also penetrating in the intradural space, surrounding the root bundles of nerve fibers, and destroying them to a great extent. In some places portions of the tuberculous masses can be traced through the intradural space to the pia-arachnoid, where they produce an inflammation of the latter membrane, so that the pia presents in certain areas thickened portions filled with leukocytes. Close observation shows this infiltration continuing into the white substance of the cord and producing the above-mentioned disintegration. The leukocytic infiltration affects the bloodvessels of the entire section of the cord. The tuberculous neoplasm presents the usual characteristic features with the well-known lymphoid tissue and areas of necrosis.

The pathological process, in order of its development, therefore, can be summarized as follows: Tuberculosis of the vertebræ, formation of tuberculous tumors extraspinal and intraspinal, the latter on the external surface of the dura; tuberculous pachymeningitis; extension of the tuberculous process through the dura to the pia-arachnoid and through the latter into the cord itself.

The interesting points to which I wish to call special attention are the following:

1. Pain was only slight through the entire course of the disease

until admission to the hospital, when it had totally disappeared. The rule is that in meningeal tumors, pain is usually of great severity. The cause of the lack of pain in my patient was probably the absence of any marked involvement of the posterior roots. The latter were seen somewhat degenerated and covered with a slightly thickened pia only on the one side of the tumor.

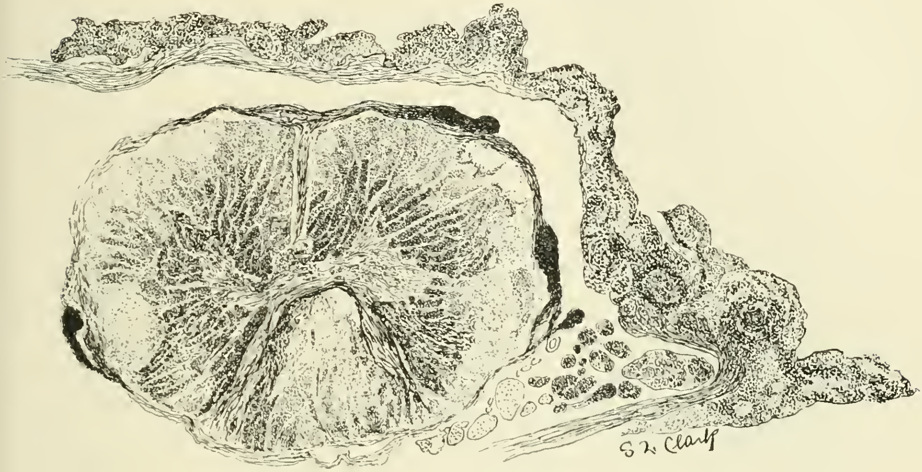

FIG. 1.—The level of greatest destruction of the spinal cord.

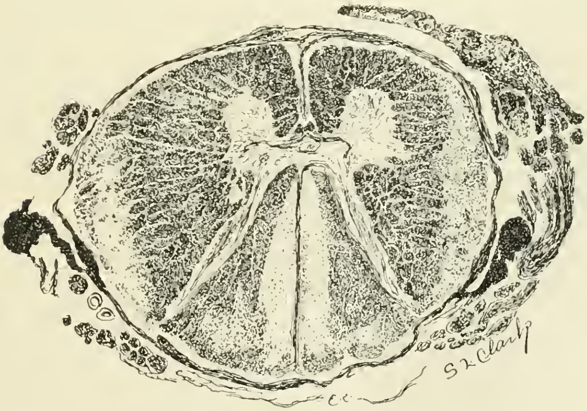

FIG. 2.—Above the level of greatest destruction of the spinal cord.

2. The tumor being extradural, places the case among the infrequent ones. Schlesinger, who made a statistical study of 400 tumors of the spinal cord, found only two extradural ones.

3. The tumor occupied the lower cervical portion, while the usual seat is the dorsal region. According to Collins' statistics of seventy cases, only fifteen were in the cervical region.

4. The extension of the pathological process could be traced through the dura and pia-arachnoid into the cord. The appearance of the sections at the level of greatest destruction of the cord tissue were those of tuberculous cords.[1] The vacuolation alongside of small masses of necrotic tissue in which is seen infiltration of round cells in the preserved small arterioles, and occasional giant cells, are all indications of tuberculous invasion of the cord itself. Although repeated attempts at staining for tubercle bacilli failed, this does not exclude the tuberculous condition of the cord tissue. Homen's[2] experiments prove conclusively that, while in recent cases the pathological changes of the nervous system depend upon the localization of the bacteria, in older cases in which the bacteria cannot be found, the alterations depend upon the poisons elaborated by the bacteria.

5. As a last interesting point, I wish to speak of the condition of the reflexes.

As is well known, a destruction of any segment of the cord may give flaccid or spastic paralysis. According to some neurologists the lumbar cord possesses an autonomous centre, so that the patellar reflex presupposes the integrity of the lumbar arc. If this arc is suddenly severed by a section of the cervical or thoracic segments, the knee-jerk disappears immediately after the shock; but later it will be reëstablished simultaneously with spasticity. The experiments of Ferrier, Sherrington, and Crocq are corroborative of this view.

Bruns, van Gehuchten, and Nonne advanced another theory. They say that in order to obtain a patellar reflex, not only the integrity of the lumbar cord, but also of certain cerebral centres is indispensable. When the lumbar centre is suddenly severed from its connection with the upper centres by a complete and rapid section of the cord, a flaccid permanent paralysis will ensue in spite of a descending degeneration of the pyramidal tract.

In chronic cases, that is, in cases with a slow and progressive destruction of a segment of the cord spastic paralysis appears at the onset and remains as such until the end. Raymond[3] calls attention to and reports two cases of a special tumor, psammoma, which is of a strictly localized nature, without tendency to generalization, and of a very slow evolution. This tumor originates in the meninges, compresses gradually a segment of the cord similarly to a solid, foreign body, and increases in size continually until complete destruction of the segment of the cord takes place. In such cases there is the best opportunity for observing a gradually oncoming and permanently remaining spasticity. Senator[4] also

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[1] See A. Gordon's case of Pott's disease, Jour. Nerv. and Ment. Dis., August, 1904.

[2] Neurol. Centralbl., 1904, No. 3, p. 21.

[3] Revue Neurol, 1902.

[4] Deut. Ztschr. f. klin. Med., 1897.

reports a case of psammoma of the cervical region, which produced a complete destruction of a cervical segment, and in which he observed spastic paralysis during the entire course of the affection.

In opposition to these facts I cite the facts of my case. The patient presented flaccidity during the entire course of her affection. Six months elapsed between the onset of the affection and death. This period is certainly more than is necessary for a descending lesion of the lateral columns to produce a spastic state of the paralysis, either by suppressing the inhibitory influence of the brain or by stimulating the cells of the anterior cornua. The destruction of the cord between the lower cervical and upper thoracic segments was, I may say, complete. The pathological process was slow and progressive.

That there is a centre in the lumbar cord, the above-mentioned experiments of Ferrier and others prove conclusively. But the question of an independent character of this centre presents certain objections. Assuming the existence of an autonomous centre in the lumbar cord, which in this case was totally severed from the encephalon, why did spasticity not develop? Is not this case an illustration of the dependency of the cord centre upon cerebral centres? I must conclude, therefore, that the reflex mechanism has multiple relations. The analysis of the accumulated observations tends more and more to show that the exclusivism of Charcot and Vulpian, on one hand, of Bastian, Crocq, Collier, and Buzzard, on the other, cannot be accepted, but that Grasset's views are more in conformity with facts—namely, that spinal, basilar, and cortical regions contain centres for reflexes and for tonicity.

SCLEROMA OF THE LARYNX.

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It is always of interest to record the phenomena of an unusual affection, and the study of scleroma of the larynx becomes the more interesting, for the reason that, like all diseases due to a bacillus, the contagious nature of the disease is to be considered, together with the possible need for recommending such action as will tend to prevent its spread.

We are all familiar with the interesting cases of scleroma of the nose, several of them having been recorded in our literature, but the case that I report herewith is the first instance of the affection beginning *de novo* in the larynx to be presented and published in North America. Like all cases seen in this country, this one had its origin in that part of Europe occupied by the Slavic race, in the northwest part of the old Kingdom of Poland, which belongs in part to Russia, to Austria, and to Prussia. The history follows:

On February 7, 1906, Annie W. presented herself at my clinic in the Throat and Ear Department of Mt. Sinai Hospital Dispensary, complaining of hoarseness and dyspnœa. The patient is a well-developed, healthy looking young woman, and gives her age as sixteen years, although from her height and weight she appears several years older. She was born in Russian-Poland in the Province of Wilna. She states that she never had any previous illness and was entirely well until three years ago, when while aboard ship, on her way to America, she caught cold and became hoarse; since that time the hoarseness has persisted, she has had masses of crusts and much secretion from her nose and nasopharynx, and within the past few months there has been increasing dyspnœa. The inspiratory effort has become more and more labored. There was at no time pain, fever, or loss of flesh. She has been able to continue her arduous duties as general house-servant. There is not now nor has there been any fœtid odor. Her parents are living, and she has brothers and sisters, all of whom are well. She can recall no one with whom she came in contact who had any similar condition or nasal disease.

Examination of the nose revealed quantities of secretion with crust formation, which, when removed, left a raw surface in places; but there was no hypertrophic spots anywhere. The nasopharynx and pharynx were absolutely normal in appearance. In the anterior commissure of the larynx there was the same crust formation, which was readily removed. There was a subglottic mass below the right cord, protruding into the larynx, diminishing its lumen by about one-half, and having a grayish appearance. The diagnosis of chronic catarrhal rhinitis and nasopharyngitis and laryngitis with a subglottic growth was made.

After thorough cleansing, portions of the mass were removed with the laryngeal forceps. They were small masses of pale tissue, translucent to a degree, resembling soft adenoid tissue. These were submitted, with the diagnosis of subglottic growth, to the Pathological Department of Mt. Sinai Hospital.

I am indebted to the chief of that department, Dr. F. S. Mandlebaum, for the report which follows:

"The specimen received for examination consists of several small pieces of a rather soft, grayish-white tissue covered with mucosa. After fixation in various solutions and hardening, they were embedded in celloidin and in paraffin. The sections were stained by the usual methods, as well as by various special stains. When viewed with the low power the sections convey the impression of a rather uniform cellular structure not unlike that presented by ordinary granulation tissue, and covered with apparently normal epithelium.

"With a higher power, several varieties of cells are seen. Granulation cells with small round nuclei are present in large numbers; also larger cells with an abundance of protoplasm. Besides these, plasma cells are found near the bloodvessels, and two varieties of larger cells to be described below. A delicate stroma of connective tissue is found throughout the sections. The bloodvessels are numerous and show, almost without exception, a marked proliferation of the adventitia. A few leukocytes are present in the neighborhood of the vessels, and the capillaries show a slight proliferation of endothelial cells.

"The distinguishing feature of the sections, however, is the presence of two varieties of cells mentioned above. The first is a huge cell with a homogeneous protoplasm which stains poorly or not at all. Its nucleus, when present, is always situated at the border of the cell. The general appearance is that of an enormous fat droplet. A very few of these cells show nothing further, but for the most part they contain bacilli. These are grouped almost without exception at the periphery of the cell body and the central part is quite free. A few of these cells are seen, however, entirely filled with bacilli. With the oil-immersion lens the bacilli are rather short and plump and their ends are rounded. Some appear almost like cocci. With some stains a faint indication of a capsule is seen. The bacilli are easily stained with the usual basic aniline dyes and also take the hematoxylin stain readily. These cells are those first described by Mikulicz as diagnostic of rhinoscleroma. They are found in great abundance in all the sections, but seem to have no relationship with any definite structure. A few bacilli are seen lying free between the cells, but almost without exception they are present only in the cells just described.

"The second type of cell is also of large size. This is the hyaline cell, which some authors consider to bear some relationship to the Mikulicz cell. Several varieties of these hyaline bodies are present, showing various stages of the formation of the hyaline substance. The form most commonly seen consists of many spherical granules quite round in shape and usually of uniform size. These fill the cell completely, and the nucleus is often seen in the exact centre of the cell, giving the appearance of a rosette. Other types show the nucleus pushed to the side, while here and there simple masses of hyaline material of a perfectly spherical form are seen lying free between the cells. With hematoxylin the hyaline bodies are tinged faintly blue, while eosin stains them intensely red. In none of the hyaline cells could any bacilli be seen. In sections stained with Unna's polychrome methylene blue, followed by aniline alum, the hyaline bodies are colored greenish blue. With Gram's method many peculiar variations are noted. Some show a flattening of the hyaline granules from pressure in the cell body, others show that certain granules stain more deeply than those immediately adjacent, while still others hardly stain at all. With Gram's method the hyaline bodies can be studied with greater ease than with any other stains employed. Although many observers disagree as to the reaction of the bacilli in cultures to Gram's stain, I find that in sections, if the decolorization is not carried too far, the bacilli hold the color when treated with this staining method. Regarding the number of hyaline cells present, as many as eighty to one hundred could be counted in one field with a medium-power objective."

In order to obtain, if possible, the bacilli in pure culture, further portions were asked for; these were removed with the customary bacteriological precautions, and from the freshly cut sterilized surfaces the bacteriological examinations were made. These examinations were conducted by Dr. H. L. Celler, who reports pure cultures of a mucoid, Gram-negative, capsulated bacillus, belonging to the Friedländer group. With the facts in hand, the presence of hyaline bodies, Mikulicz cells, and the bacilli obtained by culture, the diagnosis of scleroma (rhinoscleroma) is fully established.

I must admit that I was wholly surprised with the diagnosis presented by the pathologist, for there was nothing in the appearance of the growth that clinically resembled scleroma, and the impression is made that many other cases would be so diagnosticated if carefully sought.

The patient's condition, both as to hoarseness and dyspnœa, improved perceptibly from the first removal; bleeding was slight, and no reaction occurred. Her condition at a recent examination, three months after the removal of the growth, was of absolute freedom from dyspnœa and hoarseness; the copious catarrhal condition had greatly diminished, and there was a small grayish exudate still below the right cord.

A study of the literature of scleroma of the larynx must include those under the heading of rhinoscleroma. How vast the subject is may readily be gathered from the fact that Juffinger[1] has a list of 180 references to the general subject. Since the publication of Pieniazek's[2] article on rhinoscleroma a number of reports of cases of scleroma of the larynx have been recorded, and to enable a fuller study of the subject to the present time they are here briefly stated, reference being made only to those of undoubted authenticity and those in which the larynx was involved either alone or at first.

Our own literature on this subject is scant. Shurly[3] mentions Cornil and Alvarez to have recorded a case of scleroma confined to the larynx. Goodale[4] makes brief histological mention of rhinoscleroma. Ingals[5] says there is no proof of its contagiousness, while Kyle[6] mentions the variety beginning in the nose, extending to the pharynx and larynx, but does not speak of the affection originating in the larynx. In several instances members of the same family have had the same affection: Robertson[7] reports 2 sisters with scleroma, Secretan[8] 2 brothers, Baurowicz[9] 2 sisters and 2 brothers, Streit[10] 2 sisters, Gerber[11] 2 sisters (these latter may be the same ones mentioned by Streit). These would certainly indicate contagion.

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[1] Quoted by Mracek, Handbuch der Hautkrankheiten, Wien, 1904, iii, 567.

[2] Handbuch der Laryngologie, etc., 1898, pp. 1304–1328.

[3] Diseases of the Nose and Throat, p. 333.

[4] Diseases of the Eye, Ear, etc., Posey and Wright, p. 705.

[5] Diseases of the Eye, Ear, etc., Burnett, Ingals, Newcomb, p. 283.

While the diseased condition is usually found beneath both cords it is, as in my own case, occasionally one-sided. Thus, Juffinger,[12] in his excellent monograph on 38 cases of scleroma, says that he has been unable to find the record of any one-sided case of laryngeal scleroma; he has seen such a case, in which nothing was found in the nose and nasopharynx, but on removal of portions of the subglottic infiltration the diagnosis was established both by culture and the microscope. P. Pieniazek describes the condition and adds that Stoerk's blennorrhœa must be considered as scleroma, as also the cases described as chorditis vocalis inferior.

He formerly considered scleroma and hyperplasia of the mucous membrane as two different entities, but as the recurrences of the latter inevitably followed he reached the conclusion that they are all scleroma. He found the diseased state most often in the region of the glottis, particularly below the rima glottidis, in and over it, in the vestibule, and rarely in the trachea and bronchi. There may be diffuse infiltrations, large or small nodules, forming a granulomatous mass, protruding or lying flat on the surface of the mucous membrane. The largest tumefaction appears on the posterior tracheal wall and the small growths are soft and consist mainly of the Mikulicz cells. In time the number of round cells and connective tissue increases and the growths become firmer. Solitary nodes grow more slowly, contain more connective-tissue fibers, and the growths become very hard. They may, however, be quite soft and yielding.

As to the manner of onset: The prodromal catarrh begins in the nose and may persist for years before it reaches the larynx, and it may exist for years in the larynx before the formation of a scleromatous infiltrate appears. It is apt to be a long time before it spreads further, and hence it is rare for the bronchi or trachea to be affected early. It may last dozens of years, and often remains limited to a single spot, and it may be associated with pharyngeal and rhinoscleroma. There are cases in which, beyond the prodromal catarrh, there are no other symptoms.

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[6] Diseases of the Nose and Throat, p. 150.

[7] Internat. Centralbl. f. Laryngologie, Band viii, 70.

[8] Ann. des mal. d'oreille, du nez, etc., 1894, xx, 675.

[9] Arch. f. Laryngol. und Rhinol., 1900, x, 363

[10] Ibid., 1903, xiv, 257.

[11] Berl. klin. Woch., 1903, xl, 491.

[12] Das Sklerom der Schleimhaut der Nase, des Rachens, des Kehlkopfes und der Luft-röhr, Wien, 1892.

E. Bunzl-Federn[13] reports a case of scleroma of the upper air passages, in a male, aged nineteen years, born in Bohemia. His history is that three years before observation he began to be short of breath when walking rapidly; crust formations in the nose and throat followed. Six months before observation the dyspnœa became so great at night that he believed himself to be choking. He would then cough up hardened secretion and breathe freer. He was pale and anxious; there was stridor, dyspnœa, and loss of voice. Dark-green crusts filled the larynx and nose, which when removed relieved the dyspnœa. The cords were red and apparently broader than normal. This patient lived in Russia until he was fourteen years of age. The diagnosis was proved microscopically and bacteriologically; there is much to show that it began in the larynx.

Baumgarten[14] records a case in a female aged twenty years. For several weeks she has had two or three suffocative attacks daily, and became cyanotic. The aryepiglottic folds were infiltrated; there was slight mobility; the mucous membranes of the larynx were anemic and covered with small, white, dried blisters; the false cords and the interarytenoid folds were hypertrophied, and the true cords were hidden. The mucous membrane beneath the cords and that of the posterior wall were infiltrated and pushed forward, decidedly narrowing the lumen. Cultures from the nose and throat resulted in proof of the diagnosis. He has seen several cases primary in the larynx, but there were here and there nodules, while this patient had but the one in the nose, and this latter he believes to be of recent origin.

P. H. Gerber[15] says that no doubt the original nomenclature should be changed from rhinoscleroma to scleroma, especially if we accept subglottic cases of blennorrhœa (Stoerk) as of this variety. Without microscopic examination it would not have occurred to him to place the cases of chorditis vocalis inferior and similar ones among the scleroma cases. He records 10 cases of scleroma. In 9 subsequent cases there were no manifestations of changes in the external nose either at the time or for years after. The nose in 2 cases showed normal conditions, in 3 ozena, in 1 the usual hyperplastic rhinitis, and in 3 typical scleroma. In the posterior nares the septum was more or less thickened and the passages narrowed; in 1 case the pharyngeal tonsils were hypertrophied In none of the cases were there any typical changes in the pharynx; in 2 there were hypertrophied tonsils and granular pharyngitis. In the larynx in 3 cases the stenosis was superglottic; in the other 6 there was the usual subglottic swelling, partly with and partly without ozena of the trachea. Microscopic examination was not made in 3 cases; in 1 the result was negative; in 2 there was bacteriological examination only, while in 3 the diagnosis was confirmed histologically and bacteriologically; 4 cases came from Russian Poland and 5 from East Prussia; none of the latter had ever been in Russia.

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[13] Prag. med. Woch., March 30, 1899.

[14] Report of the Meeting of Hungarian Ear and Throat Physicians, October, 1898, Internat. Centralbl. f. Laryngologie, 1899, xv, 477.

[15] Arch. f. Laryngol. und Rhinol., 1900, x, 347.

Gerber says further that in the first series of 87 cases described by Wolkowitsch there were but 4 with normal external noses, while in Juffinger's 38 cases there were 34 with normal noses; the latter cases make the impression of milder infection. This cannot be attributed to early or late appearances, as he has had an opportunity of watching the patient from five to eight years; but it was due to a lesser virulence of the virus in all probability, and to the different geographical distribution. He is confident that the disease was conveyed from Russia; although its contagiousness is not proved, yet it has much in common with leprosy. He believes that the cases of Wolkowitsch, Juffinger, and his own show varying degrees of this infectious process; at each stage there is a diminution of the virus due perhaps to geographical conditions, commensurate with the increasing distance from the focus of the disease.

Bawrowicz, in a study of 100 cases of scleroma, says that the changes are more frequently found in the subchordal space than in the vicinity of the base of the epiglottis, and finally at the laryngeal entrance, but always on the inner surface of the larynx. The posterior wall of the larynx is rarely affected. Beginning anteriorly as a subchordal infiltration, it slowly descends to beneath the processus vocalis. This is rarely reversed. The tumefaction is at first pale red in color, then white, of smooth surface, connected either with the under surface of the cords or separated by grooves of greater or lesser depths. They are rarely one-sided, and the size of the lumen depends on their growth. The voice is usually affected. With the bacteriological or histobacteriological examination we are enabled to have a firm foundation for diagnosis and to differentiate it from lupus. In the early stages of the subchordal thickening there is a similarity to submucous inflammation, a perichondritis of the cricoid, a luetic or tuberculous infiltrate or new-growth; in primary conditions we should exclude these in the differential diagnosis. The length of time of change, slow development, absence of pain, and drying of secretion will aid in differentiating. In 6 of his cases the disorder occurred primarily in the larynx.

Additional cases have been reported by Majewski[16] (subsequent tuberculosis, death of the patient, and autopsy), Barth[17] (a subject who had never lived in a scleromatous country), Belogolow,[18] Ichemolossov[19] (involvement of the upper lids), Lenart,[20] Frankenburger[21] (involvement of the sternocleidomastoid muscle and superficial fascia, in addition to the soft palate and larynx), Ziffer,[22] Novotny,[23] Streit,[24] Hausel,[25] Deutschlander,[26] Lissauer,[27] von Zeissl,[28] von Sokolowski,[29] von Schiller,[30] Zienbicki[31] (fatal hemorrhage from erosion of the innominate artery on the fiftieth day after tracheotomy), and Petrini-Galatz[32] (the first native case in Roumania). Von Schrötter[33] also reports several additional cases that are of value in extending our knowledge of the course, pathogenesis, and extension of the disease. In 2 cases the tongue was retracted so much that a high degree of stenosis of the faucial isthmus was produced. In 1 of these cases, after lasting twenty years, the lesions seemed to remain quiescent. Another case suggests that the infection may remain latent for more than ten years, and another that the laryngeal diaphragms differ materially from the ordinary thin membranous diaphragm.

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[16] Monat. f. Ohrenh., 1900, xxiv, 250.

[17] Arch. f. Laryngol. und Rhinol., 1903, xiv, 573.

[18] Rev. hebdom. de laryngol., 1903.

Streit refers to the occurrence of the disease in Germany. He states that since 18 cases have been found in Germany (formerly it was confined to Galicia and Poland, but now may be found in Silesia and Prussia), the disease is more dangerous than leprosy, especially since leprosy is more readily diagnosticated. Having visited the scleromatous districts under governmental direction, he declares scleroma to be contagious, and he proposed: (1) Scleroma should be classed among the diseases requiring notification from the attending physician; (2) the foci should be under the particular control of the health officers of that district; (3) known cases should be seen at least once a year by the health officer, and be instructed in necessary hygiene; (4) every two or three years a special commission, one of whom at least should be a specialist, should visit the foci and make special examinations of the other members of the family; and (5) those requiring medical care should be sent at the cost of the State to Königsberg or Breslau, where a special place should be reserved for them. Private sanitaria are not available, as the segregation cannot be enforced in accordance with hygiene.

[19] Rev. hebdom. de laryngol., 1903.

[20] Internat. Centralbl. f. Laryngol., 1903, 205.

[21] Wien. klin. Rundschau, 1902, xvii, 435, 451.

[22] Wien. med. Woch., 1902, lii, 1252, 1314.

[23] Prseglad Lekarski, 1903, No. 9.

[24] Loc. cit.

[25] Internat. Centralbl. f. Laryngol., 1904, xx, 153.

[26] Ibid., xx, 616.

[27] Allg. med. Central Zeitung, Berlin, lxxii, 111.

[28] Deutsches Klinik, 1905, x, 440.

[29] Internat. Centralbl. f. Laryngol., 1905, xxi, 128.

[30] Ibid., 185.

[31] Ibid., 265.

[32] Med. Orient, Paris, 1904, viii, 219.

[33] Verhandl. der Deutschen Pathol. Gesellschaft., 1905, p. 215.

Schilling[34] reports a case, probably rhinoscleroma, associated with diphtheria bacilli. Rhinoscopically there was a tumor the size of a cherry-pit, and whitish tumefactions of uneven surface, narrowing the lumen of the larynx, but there was no dyspnœa. Smears taken at different times showed diphtheria bacilli. The clinical picture had no analogy with that of chronic diphtheria. Portions of the growth from the larynx showed characteristic scleromatous conditions, whence Schilling believed there was an original scleroma with secondary diphtheria.

The diphtheria bacillus is often found in the mouth, and the diseased mucosa might well propagate the bacillus without the symptoms of diphtheria. The lesson to be derived is, not to depend entirely on the bacteriological examination; hence removal and examination of portions of the growth are essential. If the Mikulicz cells are found, we are compelled, in the light of our present knowledge, to accept the diagnosis of scleroma. Diphtheria serum had no effect in diminishing the bacilli in Schilling's case. The patient did not come from a scleromatous country; he is said to have worked with a Galician who had some nasal disease, and he may thus have become infected.

Buschke[35] does not depend too much on the presence of the bacillus for the diagnosis, since its etiological significance lately has been made dubious.

Of other spontaneous cases of scleroma occurring in remote countries there are two in Switzerland, and Wende records a case as occurring *de novo* in the United States. The latter does not seem to have been conclusively proved histobacteriologically.

As regards etiology, scleroma occurs oftenest in people who live in filthy surroundings, in huts with the bare earth for a floor, and in close proximity to animals like cows, pigs, dogs, and cats; and yet a fair proportion of those affected belong to the better classes. It is most likely that the former are most readily subject to a catarrhal affection, and that thus a place for the ready lodgement of the disease is found. Injuries to parts may add to the susceptibility. While it affects the nose more frequently than the nasopharynx and larynx, it may affect the mouth, the ear, the lids, and the cheeks. It is indubitably proved to originate in the larynx in some cases.

The prognosis as to recovery is bad. The general health may not be impaired save from such symptoms as the varying stenosis occasions. The disease may progress or remain quiescent for years; sooner or later there is recurrence.

TREATMENT. The external conditions have been much benefited by the use of the Röntgen rays. Removal of the laryngeal scleroma endolaryngeally has at times benefited the patient, as has the pressure of an O'Dwyer tube, both before and after tracheotomy. The increasing-sized Schrötter tubes have been useful for the time being.

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[34] Arch. f. Laryngol. und Rhin., 1905, xvii, 209.

[35] Berl. klin. Woch., 1905, xlii, 270.

From a study of this subject I am led to conclude that scleroma is a chronic incurable affection, contagious to a certain degree. It is safe to assume that there are many more cases than even the large number already recorded. As the highest art of medicine lies in prevention and not in treatment, it seems advisable that the sanitary authorities take appropriate action now, while the cases are comparatively few and far between. Whilst not agreeing with Streit as to the relatively greater dangers in scleroma than in leprosy, yet, because it is easier to control the few cases now than the many the future is sure to bring, his views as to sanitary regulations in Germany and the territory adjacent to the infectious district should receive cordial endorsement. As far as our own country is concerned every quarantine officer should look upon undoubted cases of rhinoscleroma as infectious, and the subjects thereof should not be allowed to land, but be deported at once. In addition, every case of longstanding hoarseness, with or without dyspnœa, especially when associated with chronic catarrhal conditions, should be detained until such time as some expert authority can determine the presence or absence of scleroma.

THE HEREDITY OF FORM AS ILLUSTRATED IN PATHOLOGY BY A STUDY OF THE CYSTS OF THE MIDDLE TURBINATED BONE.

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Some ten years ago I had occasion to comment[1] on the pathology and pathogeny of bony nasal cysts. Even at that time I found, on reference to the literature, that conclusions at which I had arrived from independent study had been anticipated in the works of other observers. The special literature of the subject previous to the above reference is given in an article by Stieda.[2] Notwithstanding the large amount of testimony to the correctness of the views of Stieda, Glasmacher, and others, the text-books on rhinology and many of the special articles which have appeared in the last ten years have contained a surprising number of fantastic and erroneous ideas of the growth of these bone cavities. These I shall not review.

[1] New York Med. Jour., June 27, 1896.

[2] Arch. f. Laryngol., Band iii, S. 359.

I have learned, from experience with the literature of the pathogenesis of nasal polyps, how difficult it is for even the most important and the clearest of demonstrable evidence to make headway against erroneous pathological prepossessions in rhinology. After a time, however, it prevails. Neither shall I give here any but the briefest account of what may be found in the sources I have indicated.

In about 70 per cent. of the cases the clinical history is as follows: A woman from twenty-five to forty-five years of age complains of long-existing and perhaps still increasing nasal obstruction on one side, less frequently on both sides. This may have caused nasal deformity and perhaps some pain from bony pressure within the nose. Examination shows a smooth, globular mass springing from the middle turbinated bone, pressing upon, indenting, or displacing the nasal septum. It may be hidden by an œdematous polyp, or the mucosa may be atrophic. It may be of such a configuration that it can be removed entirely, with more or less of the middle turbinated structure with which it is seen to be incorporated, or it may be possible only to remove it piecemeal with bone forceps. It does not recur.

Usually sections are made in such a way that on microscopic examination channels of communication with the cavities in the body of the ethmoid bone, of which the middle turbinate is a process, cannot be detected. Usually such a channel exists. Its absence can only be satisfactorily demonstrated by careful serial sections, including the entire spherical surface of the bony walls. This I have never succeeded in doing, and I do not know if it has been done with a technique free from criticism. The matter, however, is not of importance,[3] because on the understanding that the primitive cavity may have been either a diverticulum communicating through an ethmoidal cell with the surface, or a cell in the bone shut off during the development of embryonic or adolescent life from other cells of the ethmoid, the explanation of the findings in the contents is identical. The most important of these is that the bony walls are lined by columnar epithelium loosely attached to a thin periosteum by scanty strands of connective tissue. Sometimes this is destroyed by a purulent inflammation or by mucoid degeneration, and the pus or glairy mucus will flow from the ruptured walls.

Even from the typical history thus far it is clear that the genesis of these cases is essentially the dilatation of a preëxisting cell within the body of the turbinated process, which is the analogue and of similar embryonic origin with the cells in the lateral bodies of the ethmoid bone. Whether these are closed cysts or still diverticula in the adult, the presence of an epithelium is indicative of their communication with the surface at some time of adolescent or embryonic life.

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[3] I might have added that the only importance of noting that usually these cavities communicate with other cavities and through these with the exterior lies in the negation that it gives to the idea that these cysts are due to pressure of exudates within. This has frequently appeared to me as invoking a cause for a cyst which does not suffice and only obscures the true pathogeny.

Many years ago Kölliker[4] asserted that not only the Howship lacunæ and the medullary canals of the long bones, but also the accessory sinuses of the nose were hollowed out by the action of the osteoclasts or bone-absorbing elements, and the inner wall of these nasal cavities was the best locality in which to study them.

Fig. 1 shows a section through the bony wall of the maxillary antrum of an infant. Under the microscope the appearances are as shown, except that the cellular detail has been omitted. The worm-eaten inner edge, with a clump of lime-salts detached by the shrinkage of a giant multinuclear cell which the decalcifying reagents have obliterated, and the smooth outer edge continuous with the periosteal and submucous fibrous tissue show the process at a glance. The conditions in Fig. 2 are hardly less convincing, showing the same evidence of the action of the osteoclasts on the inner wall, and of the osteoblasts on the outer wall, of a bony cyst of the middle turbinate.

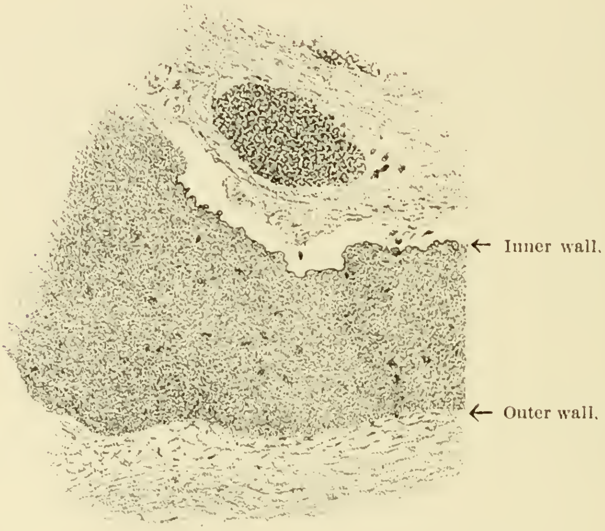

FIG. I.—Wall of an infant's maxillary sinus, showing lime salts taken from the inner wall, thus enlarging the cavity in the bone. (Camera lucida drawing. × 600.)

There is still discussion as to the origin of the osteoblasts or bone-forming cells, but there is no reason to dissent from the generally accepted view that both pathologically and physiologically they are derived from the fibroblasts, which are themselves derivatives of the plasma cells and the lymphocytes.[5] There is very much less unanimity as to the origin of the multinuclear giant cell first found by Robin in the marrow of the long bones and called a myeloplax, but renamed later by Kölliker an osteoclast. The latter author[6] says they are derived from the osteoblasts. Ebner, who in 1902 revised Kölliker's classical work, adding a third volume, declares that it has been definitely proved by J. Schæffer that they are derived from the endothelium of the capillaries by a modification of the cytoplasm and a fusion of the cell bodies into one, enclosing a number of nuclei. In Fig. 3 is to be seen a row of osteoblasts which have evidently been transformed from fibroblasts.[7] In Fig. 4 will be seen transitional forms in the Howship lacunæ, a grouping of nuclei to form a giant cell or osteoclast where the bone is becoming absorbed and porous. The common cell body cannot

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[4] Handbuch der Gewebelehre des Menschen, 6te Auflage, Band i, S. 354.

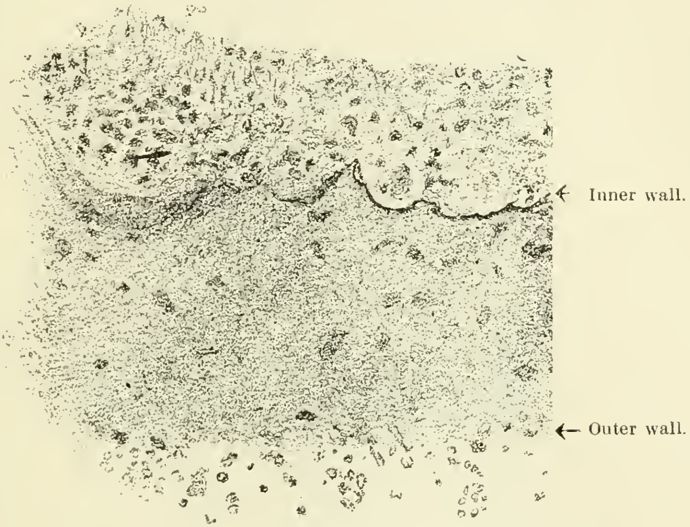

FIG. 2.—Section of the wall of a cyst in a woman's middle turbinated bone, showing in outline the inner wall eaten away by the action of osteoclasts. (Camera lucida drawing. × 600.)

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[5] It is not my purpose to review the voluminous literature of this subject, but reference may be made to the recent contributions of Sormani, Virchow's Archiv, 1906, Band clxxxiv, Heft 2; Maximson, Ziegler's Beiträge zur pathol. Anat., 1906, Band xxxiv, Heft 2; Bidder, Archiv. f. mikroskop. Anat., 1906, Band lxviii, Heft 1; Poscharissky, Ziegler's Beiträge zur pathol. Anat., 1905, Band xxxviii, Heft 1; Goetsch, ibid., 1906, Band xxxix, Heft 2.

[6] Loc. cit., p. 347.

[7] For Figs. 3 and 4 I have made use of a fibroma springing from the periosteum of the inner border of the alveolus of the upper jaw which contained some very small areas of newly formed true bone. Not only was a very active process going on here, but decalcification was easily accomplished and resulted with less damage to cellular structure than usually occurs with older and firmer specimens. The process is exactly the same in bone lesions of the middle turbinate and in the dilating walls of the accessory cavities of the nose in infants, as in this neoplasm. Hematoxylon and eosin stain.

here be distinctly made out, but the process is unmistakable, while elsewhere in the specimen can be seen a few typical osteoclasts. Fig. 5 shows a fully developed osteoclast along the inner border of the wall of the bony cyst of the middle turbinate shown in Fig. 2. The artist has rather heightened the outline of the cell body and brought the whole cell into more prominence than is usually seen, since they easily disintegrate under the use of decalcifying agents. It is true such appearances are deceptive and need the support of coincidence with a theory of broader significance. This is the point of biological interest and should furnish an excuse for the detail of much which has preceded that belongs to elementary histology.

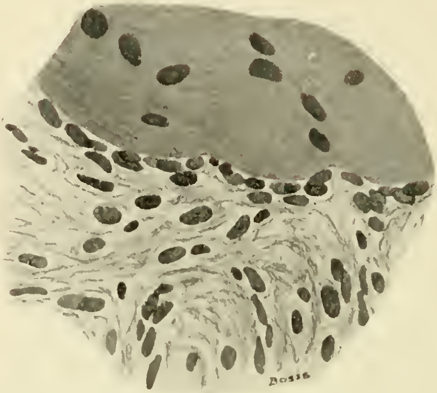

FIG. 3.—A row of osteoblasts derived from the fibroblasts of an epulis of the upper jaw. (1/12 oil immersion lens.)

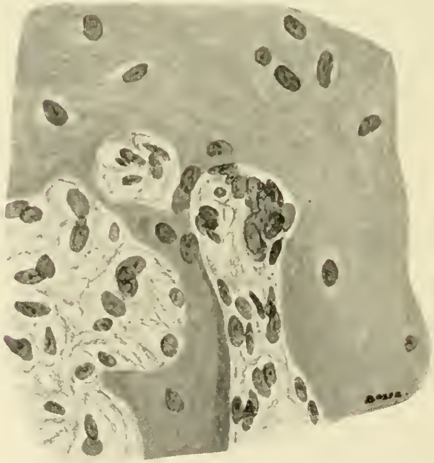

FIG. 4.—The transition stage from osteoblasts to osteoclasts. Same case as Fig. 3. (1/12 oil immersion lens.)

John Hunter in his day clearly saw that the animal bony frame took its shape through a process of resorption. Julius Wolff[8] has clearly demonstrated, acknowledging his indebtedness to Culmann, how this architectural construction in the long bones, even in pathological cases, is an answer to the strain and stress put upon them by the weight of the body and the influence of its muscles, and is in exact and economical correspondence with that demand.

The universal conception, now familiar to us in biology as well as in physics, of equilibrium is useful in understanding the interrelation of osteoblast and osteoclast, and the conversion of one into the other to maintain the form of the bony skeleton. The constant demand of the system for economy of effort and material is the influence which removes such lime-salts as are not needed through the activity of the osteoclasts. The local demand for bony support, answered by the osteoblasts, is the opposing force. The resulting form of the bone is the expression of the equilibrium between the two. Wilhelm Roux in his voluminous symposium[9] has shown the wide biological application of the principles here involved. They are the very root of what we must know to understand the laws of heredity, of adaptation, and of variation.

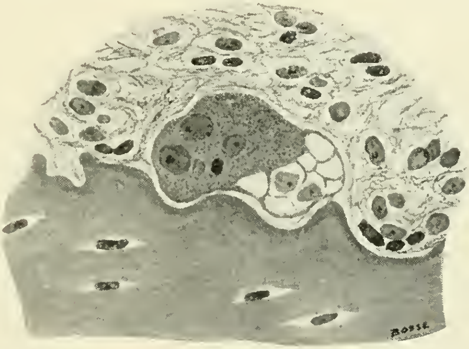

FIG. 5.—Section of the wall of a cyst in a middle turbinated bone, showing fully formed osteoclasts absorbing the inner wall. (1/12 immersion lens.)

If we are permitted to believe that the osteoclast, or bone-absorbing organ, is directly derived from the osteoblast, or bone-building organ, and that this change in morphology is an expression of a change in function, we readily perceive how plastic an instrument of heredity it is. In answer to some influence or demand, which is the mysterious and perhaps unknowable primal fount of life itself, the nuclei of the osteoblasts cluster together and their cell bodies become fused into one cytoplasm. There are appearances, faint outlines of the original cell bodies, which may be seen within the common cytoplasm, which lend an objective support to this subjective view of the question. The giant cells of the marrow may indeed be formed in the same way from the leukocytes, or the giant cells of tubercle from the endothelium of the dissolving capillaries, and I am inclined to think this also has an objective support in microscopic observation, but the giant bone cells are in all probability the ultimate product of fibroblastic and osteoblastic metamorphosis.[10] It is said the osteoclasts may again revert to osteoblasts, but I am not familiar with any evidence to that effect. Indeed, in spite of the generally accepted view, I doubt if even cartilage cells revert to fibroblasts and osteoblasts. It seems likely they also are the ultimate stage of a metamorphosis from the polyblasts and that fresh fibroblasts taking their place, as they degenerate and disintegrate, are themselves converted into bone-forming cells. While Bidder and some others declare that pathological bone processes differ from the physiological, this is not the usually accepted opinion. The hyperemia of inflammation sets these metamorphoses going anew after the physiological stimuli of growth have ceased. Professor Reinke[11] has shown in a recent publication that the pressure of the lymph due to hyperemia, either local or general, is the proximate cause alike of physiological and pathological growth and of regeneration of lost parts. He insists that the word "inflammation," as an expression of hyperemia and exaggerated lymph pressure in pathological cases, often gives rise to mistaken impressions, and he suggests the word "blastose" as covering the pathological as well as the physiological meaning; but the multiplication of terminology is always to be deprecated. Modification of conception is easier and gives rise to much less confusion.

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[8] Das Gesetz der Transformation der Knochen, 1892.

[9] Gesammelte Abhandlungen über Entwickelungs Mechanik der Organismen, 1895.

The formation of giant cells is so frequently associated with degeneration in pathological changes that we may entertain the belief there is some way, in common with the physiological, by which necrobiosis of protoplasm changes tissue-forming cells, the polyblasts into tissue-absorbing cells or giant cells. The absorption by the osteoclasts of bone salts, no longer needed on the inner walls of the accessory nasal sinuses, may in this way be of secondary significance, the primary change being the disintegration of the bone salts. This in its turn must depend on the decline in the lymph pressure, as pointed out by Reinke. The circulation of blood and lymph in the periosteum is there, as elsewhere, regulated by sympathetic nerve action. Now we have arrived at a factor whose equilibrium may be altered either locally (by inflammation) or by the mysterious hereditary forces which preside over growth.

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[10] It is very probable that the frequent occurrence of giant cells in sarcomas sprung from periosteal tissues is the expression of an heredity in the growth of the perverted tissue. Whatever may be found to be the causative stimulus, I think we must look upon malignant growths, not as misplaced tissue (Cohnheim, Ribbert), but as a heterogenesis set going by some abnormal shock to the nuclei of cells which have, as Bashford insists, entered upon their old age.

[11] Archiv f. mikr. Anat., 1906, Band lxviii, Heft 2.

That bone may be absorbed in pathological cases by other than giant cells admits of no doubt, and recently has been beautifully shown by Goetsch, while the heteroplastic origin of bone in the arachnoid, in the arteries,[12] in the lungs,[13] in the uterus,[14] in the lymph nodes and the tonsils,[15] and in many tumors of fibroblastic origin is well known, but does not invalidate in any way, but rather supports the theory of the vicarious function and the metamorphoses of the fibroblast. If, then, the osteoblasts and osteoclasts are the modelling clay of the animal frame, what of the force that shapes it?

When we study the pathogenesis of bony or cartilaginous spurs of the nasal septum in the clinic and under the microscope, occurring chiefly as they do in adult life, we are driven to the conclusion that they are hyperplasias set up, first, as a process of repair after injury, and, second, as the result of the involvement of the perichondrium or the periosteum in the inflammations of the mucosa. But there is no tendency here to a location of osteoclasts on one surface and osteoblasts on the other surface. Beneath the mucosa of the ethmoid region, however, in the formation of these bony cysts we plainly perceive the same stimuli to proliferation, a spread of inflammation from the mucosæ. Whence, then, arises the differentiation of form in the two pathological conditions, the bony spur in the one case and the bony cyst in the other? There seems no escape from the conclusion that the fibroblast has one kind of heredity in one place and another kind of heredity in another.

I have in another series of articles attempted to point out how one locality, such as the tonsils, may have a local heredity as regards the permeability to germs of the epithelium which cover them, which may not apply to another locality, because a special need exists at the spot where the air current and the food current, all that goes into the body, meet.

Whether these kindred phenomena, or rather this common phenomenon, arises from adaptation or natural selection is an entirely secondary consideration. We may readily understand how to an aquatic animal, and especially to an amphibious animal, it would be a matter of vital importance, in a survival sense, whether the osteoclasts had made his head of the proper buoyancy by establishing air spaces in it. I fancy it is in this light we must view the long-disputed meaning of the existence of the accessory sinuses—in other words, their function. The demand for lightness and buoyancy, especially in the amphibious part of our phylogeny, and the economy of nature when dealing with the individual organism, are the factors in evolution which have hollowed out these cavities. I want to point out here, also, in this study of a pathological nasal cyst, how out of the abnormal, as Burbank urges—how out of an inflammation, a variation toward the buoyancy of the animal's head in water might arise. By the survival of individuals with this advantageous disease, a modification in the race, or a new species sprung from a pathological process, is conceivable.

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[12] Rohmer, Virchow's Archiv, No. 166, p. 13.

[13] Pollack, ibid., No. 165, p. 129.

[14] Meyer, ibid., No. 167, p. 81.

[15] Lubarsch, Rückert, ibid., Bd. clxxvii.

Reverting to the subject already broached, I desire to insist that the same law which governs the local differentiation in the action of the osteoblast of the ethmoid and the osteoblast of the nasal septum governs the differentiation in the local immunity, the permeability of the surface epithelium to germs on the faucial and pharyngeal tonsils.[16] If there is evidence of this differentiated local heredity in the nasal fibroblasts, indistinguishable in form, to doubt that the proper kind of a tonsillar epithelium was evolved in the same way is to indulge in illogical mental gymnastics, and yet theories of infection are being built up by a study whose chief experimental support is derived from the behavior of the animal organism under conditions with which it has had no evolutionary experience. The peritoneal endothelium and the lymph which bathes it cannot be expected to have acquired the heredity of the tonsillar epithelium, nor can the tonsillar epithelium be supposed to have acquired an experience with the enormous numbers and the unusual varieties of bacteria used in experiments.

Having gone over a good deal of well-known ground in order to place the genesis of these bony nasal cysts in a light which may be of use in more original lines of work in biology, I also desire to draw attention to a fact which has received entirely too little consideration from the clinical observation of rhinologists. I mean the influence of sex on the development of nasal disease. It will be found in studying the records of these bony cysts that they have nearly all occurred in woman.[17] About the same preponderance in the female exists for atrophic ozenatous rhinitis. Future investigation will probably show the significance of the coincidence that both of these are essentially a bone disease, involving the absorption of bone. At present I am entirely unable to make a suggestion toward its elucidation.

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[16] See New York Med. Jour., January 6 and 20, 1906.

[17] It will be understood there are other bony cysts of the nose and its accessory sinuses whose pathogenesis and clinical history are of another nature. I cannot do better than refer the reader, for these, to Dr. A. Logan Turner's monograph in the Edinburgh Medical Journal, November and December, 1903, January, 1904.

CONCERNING THE OCCURRENCE OF NEOPLASMS IN WILD MAMMALS, WITH THE REPORT OF A CASE OF SARCOMA OF THE OVARY IN NYCTEREUTES ALBUS.

By Harlow Brooks, M.D.,

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True neoplasms in wild mammals which live under conditions that closely approximate the natural are extremely rare. This is true of relatively pure and uncontaminated species.

I am aware that this statement will not meet with the immediate approval of the general zoölogist, on the one hand, or that of the pathologist of strictly laboratory experience, on the other, since a considerable number of animals suffering from tumors appear to these classes. Zoölogists are very likely to draw their conclusions entirely from the gross appearance of simple enlargements or tumors in the animals which come under their observation either in the field or park, or at the dissecting table. The statistics so derived will lead, I believe, to conclusions quite contrary to that with which I have introduced this article, but collected in this manner, they have no bearing on the occurrence of true neoplasms present in animals living under normal conditions.

My explanation of this discrepancy in observation lies in the following facts: It may be first accepted as axiomatic that histological diagnosis only is conclusive; most of the growths reported in wild animals are unquestionably inflammatory in nature, and these can be differentiated only by the use of the microscope. Very few of the specimens seen in the ordinary zoölogical collection are kept under conditions even approximating the normal. Animals accustomed to an abundance of air and sunlight, two of the most essential elements of hygiene for nearly all mammalian life, are usually placed in quarters largely devoid of these primitive requirements. The quality and quantity of food furnished generally leaves much to be desired as a substitute for that natural to the animal, while close contact with others of the same or different species favors the inception of the numerous infectious diseases which either kill or debilitate. A further strongly depressing factor dominant in most collections lies in the small amount of exercise which the animals take, and animals, like men, are very prone to physical indolence when a strong incentive for physical exertion is lacking. All these conditions so greatly alter both the physical and the mental attitude of wild animals in captivity, a fact well recognized by animal artists of note, that I consider it self-evident that such collections can never be utilized for statistical purposes for any data supposedly based on natural tendencies.

All these effects of ordinary captivity and others, some of which are potent even in the wild life, facilitate the formation and dissemination of tumors of parasitic or vegetable origin. As examples, we may cite the frequent occurrence of hyatid and coccidia cysts and of the granulomas following tuberculous, actinomycotic, and other bacterial infections. Thus, many infective and other agents are at work which may cause the formation of tumors which the zoölogist, untrained in pathological histology, may easily and honestly mistake for true neoplasms. It is my belief that similar sources of error exist in regard to the occurrence of neoplasms among domestic animals, although I cannot state this from personal knowledge.

Pathologists of strictly laboratory experience, whose acquaintance with animals is chiefly or exclusively confined to such domestic animals as the horse, sheep, cow, dog, and cat, or to the semi-domesticated animals of the laboratory, notably guinea-pigs, rabbits, and mice, are too likely to base their opinions on the rate of neoplasm occurrence among these animals, a very erroneous conception, since all of these show the marked effects of close inbreeding. For the most part, they live under more or less artificial conditions and crossing of species is the rule rather than the exception. Furthermore, these animals, as is the case with man, though in a much greater degree, are removed more or less from the natural and beneficient effects of the law of the survival of the fittest. In short, these animals live under conditions very closely simulating those under which urban man lives, and with, as I believe, very much the same effect as regards tumor formation; as might be expected, neoplasms are not uncommon among them.

It has, therefore, occurred to me that statistics regarding the presence of true tumors among animals of the New York Zoölogical Park may be of value, since we are here attempting to keep wild animals under conditions as nearly approaching the normal as earnest endeavor and scientific ingenuity can devise with the obligatory materials at hand.

It may be argued that statistics founded on this collection are not permissible, since all diseased animals would be excluded by the thorough veterinary inspection which precedes the admission of any members to this group. This might seem a legitimate conclusion as regard superficial growths, were it not a fact that thus far (five years) no animals have been thus excluded for new growth formation. A very considerable percentage of animals received at the park are rather recently in captivity, they are carefully selected by those versed in such lore as typical representatives of their species, and for these reasons appear to me to furnish the most certain data on which we may base conclusions in regard to the occurrence rate of neoplasms in wild animals.

In the 2647 mammals under our charge for the past five years, but a single case of true neoplasm has occurred. 744 of these animals have come to complete autopsy without a single new-growth being found, with the exception of the case already cited.

Added to these cases I have a considerable series of animals secured and dissected in the field either by myself or by other physicians, most of whom happen to have been either pathologists or surgeons, and, therefore, particularly well qualified to judge. Not a single case of neoplasm has been found among these animals, which have been, however, chiefly limited to the various North American wild ruminants. Conversation with guides and pioneers has further convinced me that not only are true neoplasms practically unknown, but also that the various parasitic tumors relatively frequent in wild animals in captivity are extremely rare in those in the wild.

In view of this great rarity of neoplasms among wild mammals it seems to me particularly necessary that we study in some detail the single occurrence which has come to our notice from this considerable amount of material.

The animal was a white raccoon dog, which had been a member of the park collection for over two years before it sickened and died. It had been captured in northern Japan and has been described by Director Hornaday[1] as a new species, Nyctereutes albus.

Postmortem examination of the animal by Dr. Reid Blair, resident pathologist, showed it to be generally emaciated. All the viscera exhibited the effects of malnutrition and anemia consequent upon neoplastic disease. The lower two-thirds of the pelvic and abdominal cavity was occupied by a large retroperitoneal mass to which the coils of intestine were adherent in many places, causing a partial intestinal obstruction, notably where the entire gut had become more or less surrounded by the new-growth.

The tumor had apparently originated in the left ovary. It was well supplied with large nutrient vessels derived, not only from its original ovarian supply, but also from the trunk of mesentery and other peritoneal stems. The general color of the growth was light pink, shading off into pinkish gray. It was dense and elastic in consistence and showed a few cystic areas enclosing vesicles of clear, semigelatinous fluid. Gross diagnosis of myxosarcoma of the ovary was made, and was subsequently substantiated by microscopic examination.

Microscopic Examination. The greater bulk of the tumor is made of a stroma of delicate fibrils which apparently are almost exclusively derived from processes of embryonal connective-tissue cells. These cells are polymorphic, and contain large nuclei, many of which show karyokinetic-like arrangements of the chromatin. In a few places an indefinite stroma of adult connective tissue is made out, clearly having evolved from the mucoid tissue already described. In many places a hydremic condition is present, both cells and fibers appearing macerated, and large spaces are seen probably filled, in the fresh state, by serum. Bloodvessels are numerous; many of them are newly formed and of large size. Their walls in many areas are directly made up of the myxomatous cells and some of the larger trunks have so thin and imperfect a limitation that extravasation of red and white cells into the surrounding tissue has taken place. In addition, certain vessels are surrounded by zones of small round-cell infiltration, evidently inflammatory. Plasma cells and neutrophilic and eosinophilic leukocytes are also occasionally found, evidently, also, indicative of inflammatory reaction.

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[1] Report of the New York Zoölogical Society, 1903, p. 71.

In one place, evidently where the growth originated, probably from the ovarian cortex, large polymorphic epitheloid cells are found, arranged in alveoli in some cases, and bound together by a fairly well-defined hyperplastic connective-tissue reticulum. The cytoplasm of these cells is highly granular and in some cases intensely eosinophilic. Other cells show many vacuoles and a few enclosed hematogenous pigment granules. The nuclei are mostly large, and karyokinetic figures are frequent. Apparent transition stages of these cells into those of fibrous tissue, which is found in a quite dense capsular arrangement, can be made out, and they seem, therefore, to be of mesodermal origin, notwithstanding their epithelioid appearance. Bloodvessels are less numerous in this portion of the tumor, and they are, as a rule, more typical in structure.

Great interest lies in the discovery of this neoplasm in this particular animal, not only on account of the great rarity of the tumor, but chiefly because it occurred as the only example of tumor formation in an animal of questionable purity of species. Notwithstanding the fact that so excellent an authority as Hornaday has described this animal as a new and distinct species, we must, in the light of the great rarity of neoplasms in animals of pure species and their relative frequent appearance among those of mixed derivation, consider at least the opinion of other zoölogists who do not look upon the white raccoon dog as a pure species, but rather as a "sport," albino, or the result of mixed parentage.

The bearing of these observations on the general question of the etiology of tumors would seem to tend to substantiate those theories attributing neoplastic formations to perversions of cell growth, a tendency well-recognized to be more frequent in animals or people of imperfect development or of mixed races, or such as live under more or less artificial environment.

That true tumors are relatively rare in wild animals, particularly in those of gregarious tendencies, as wapeti, caribou, or antelope, would also, in a measure, disprove the theory of the infectious origin of new-growths or their complete dependence upon such extraneous causes as parasitic infection or peculiarities in food, drink, or habitat.

REVIEWS.

A Treatise on Orthopedic Surgery. By Royal Whitman, M.D., Clinical Lecturer and Instructor in Orthopedic Surgery in the College of Physicians and Surgeons of Columbia University, New York. Third edition. Pp. 871. Philadelphia and New York: Lea Brothers & Co., 1907.

The value of Whitman's *Orthopedic Surgery* is attested by the fact that the third edition is published within a few years of the issuance of the first. Each new edition has claimed the undivided attention of the author, and this has led to careful revision and additions, in keeping with the advances made in this special department of surgery—orthopedics. In this new (third) edition all subjects are treated in a scientific, plain, and clear style, and several new chapters are presented. The new subjects discussed are cervical ribs, iodoform filling in bone cavities, scapular crepitus, fractures of the neck of the femur in adults, and muscle transplantation. Cervical ribs, which of late have attracted considerable attention, are thoroughly discussed. The same may be said of iodoform plugging in tuberculous bone cavities. The author recommends the same treatment in fractures of the neck of the femur in adults as he uses in the treatment of fractures of the neck of the femur, or epiphyseal separation, in childhood, namely, forcible abduction and fixation, with the idea of restoring symmetry. This subject has engaged the attention of the author for some years and his convictions are well presented. Hoffa's operation for paralysis of the deltoid muscle, consisting in the transplantation of the trapezius muscle, and of the sartorius muscle in paralysis of the quadriceps extensor muscle, is well described. The text on acute non-tuberculous osteomyelitis is thoroughly revised, and the investigations of various authorities carefully considered and noted.

The volume of the text has been increased by nineteen pages, and the illustrations by forty-seven. The arrangement of the subject matter and the illustrations are excellent. Altogether the book represents the most up-to-date orthopedic ideas; it will prove a most valuable addition to the library, not only of the orthopedic specialist, but also of the general practitioner, and it cannot be too highly commended. J. A. K.

A Treatise on Surgery. By George Ryerson Fowler, M.D. Vol. I, pp. 722 with 398 text illustrations and 4 plates; Vol. II, pp. 714 with 888 text illustrations and 4 plates. Philadelphia and London: W. B. Saunders & Co., 1906.

This treatise upon surgery, fortunately completed before the untimely death of its brilliant author, is an interesting one to review and to be heartily commended as one of the best text-books of its size written in recent years. The volumes have evidently been subjected to close scrutiny by the author, and consequently the text is quite free from the many misstatements about the scientific aspect of surgery so common in most text-books. As stated in the preface, the grouping of the topics differs somewhat from the arrangement usually employed, having more of an anatomical basis. In the first section, wounds, inflammation, and fever are discussed in an interesting and scientific manner, the regard for modern physiology and pathology being markedly greater than in other text-books of this size. Injuries and diseases of the skin, subcutaneous tissues, the bloodvessels, lymphatics, nerves, fascia, muscles, tendons, bones, and joints are considered in the next section in an accurate but very brief manner. The open method of treatment for burns is not mentioned, the description of the method of repair of bone is rather inaccurate, and but twenty lines are devoted to the important subject of sarcoma of bone, with no mention of the cysts or giant-cell central sarcomas. The importance of this omission is apparent when reading further (p. 370) that amputation or disarticulation of the limb must be performed for sarcoma of bone. The section on gunshot injuries is illustrated by photographs of wounds inflicted during the Russo-Japanese War. Active and passive motion with massage, etc., are advised in the treatment of osteo-arthritis without referring to the varieties of this disease, the hypertrophic form of which should not be so treated.

The various methods of laboratory procedure as aids in surgical diagnosis are fully discussed in a separate section, and the reviewer is glad to note that the results of the frozen-section method of diagnosis are described as frequently unsatisfactory or meaningless. The dangers of operation, postoperative complications, methods of anesthesia, and the various minor surgical procedures are described in an entertaining and instructive manner in the next section. The physiology of shock, however, deserves more than the brief notice given it. Part II of the volume, regional surgery, consists of a discussion of the injuries and diseases, with their treatment, of the head, neck, and thorax. These sections are complete, although brief, the minor operations, harelip, for example, being particularly well described.

The second volume opens with the surgery of the dorsal and lumbar vertebræ, and discusses, in addition, the surgery of the abdominal and pelvic regions, the female pelvic organs, and the upper and lower extremities. The exposition of the methods of suture of the intestines and the section upon hernia are particularly good and the illustrations very expressive. Fractures, dislocations, and other injuries and diseases of the bones and joints are individually considered and seem to be excellent and modern. Certain omissions and criticisms must be noted, notably the absence of any mention of Finney's operation of gastroduodenostomy; the inadequate description of the most frequently performed operation upon the stomach, posterior gastro-enterostomy, the illustration giving the impression that a long loop is used; the omission of the Ochsner treatment for appendicitis; hypernephroma, the most common kidney tumor in the adult, is vaguely referred to as an adrenal sarcoma; Bevan's operation for undescended testicle is not considered. The section upon disease of the gall-bladder and bile ducts and that upon carcinoma of the rectum are somewhat confusing in the description of the pathology. The irrigation method of treatment is advised for gonorrhea, perineal prostatectomy is preferred, and the use of lateral traction for fracture of the neck of the femur is not mentioned.

These criticisms, together with the fact that the symptomatology of the various diseases is slighted in many places, do not retract from the value of the work. It is eminently practical, and tells in a brief, concise manner just what to do, not only for the major lesions, but also for many of the lesser affections which are so often omitted in text-books of this size. The illustrations throughout are well executed and instructive, although it is sometimes startling to see the cheerful expression of persons with portions of their anatomy laid open. G. P. M.

TRAVAUX DE CHIRURGIE ANATOMO-CLINIQUE. TROISIEME SERIE. CHIRURGIE DE L'INTESTIN. By HENRI HARTMANN. Pp. 453. Paris: G. Steinheil, 1907.

HARTMANN, the most distinguished of the pupils of Terrier, presents herewith to the surgical world the third series of his *Travaux de Chirurgie Anatomo-clinique*. He has been assisted in the preparation of the present volume by his pupils Lecène and Okinczyc—the former already well known to readers of current French literature, and the latter giving promise of further renown by his very able studies in the treatise now before us. The volume contains: (1) Statistics of the service civiale (Hartmann); (2) intestinal and gastro-intestinal anastomoses (Hartmann); (3) exclusion of the intestine (Hartmann); (4) surgical anatomy of the colon (Okinczyc);

(5) pathological anatomy of tumors of the colon (Okinczyc); (6) clinical study of tumors of the colon (Okinczyc); (7) surgical treatment of cancer of the colon (Hartmann); and (8) sarcoma of the small intestine (Lecène).

It is thus seen that although far from being a complete and systematic treatise on the surgery of the intestine, the work nevertheless covers the most important subjects under this heading, and well justifies its modest title of anatomo-clinical studies. The object sought—of combining clinical work with studies in pathological anatomy—has been fully attained, and the result, we think, cannot but impress the judicious student with the fact that a clinical study so made is after all the most truly scientific.

The most interest naturally attaches to the work of Hartmann himself. In the section on intestinal anastomoses he treats of the principles which govern the technique in such operations. As those who have seen him operate will remember, he makes much of fixing his continuous sutures by knotting them, from time to time, as they pass around the anastomotic opening. This is only one instance which exemplifies his intense care in operating—nothing is too trivial to observe, if it will, in any way, render the operation more safe; and he is rightly of the opinion that it is better to be safe than brilliant.

In the treatise on exclusion of the intestine the question is raised as to the limitations of this term. Should it include lateral anteroanastomosis for obstruction, a form of operation originally proposed by Maisonneuve as an improvement over enterotomy, usually known by Nélaton's name? Thinking that this is too wide an application of the term, Hartmann concludes that it should be limited to cases in which the intestinal canal is interrupted by one or two sections, its continuity being otherwise provided for; and that the term enteroanastomosis (short-circuiting) should be reserved for Maisonneuve's operation. As to the choice between the two operations, Hartmann is of the opinion that when stenosis of the lumen from a neoplasm is the only indication, enteroanastomosis should be preferred, because exclusion will prolong life no more, and because incomplete exclusion (section and closure of the bowel above the tumor alone) would be likely to result in reflux into the excluded loop, while complete exclusion (section and closure of the bowel both above and below the tumor) would necessitate fistulization of the excluded loop to prevent its distention and rupture from accumulated secretions. If stenosis from chronic inflammation (tuberculous or not) is the indication, he prefers complete exclusion, with fistulization of both ends of the excluded loop, to allow of topical treatment.

The articles by Okinczyc on the surgery of the colon occupy by far the greatest portion of the volume (267 pages in all). Space forbids discussing them in full. The most important point brought out in the chapter on the surgical anatomy is the existence (theoretically known to all, but practically utilized by few) of fascias of adhesion between the ascending and descending colons and the corresponding portions of the primitive parietal peritoneum. We have become accustomed to the "mobilization of the duodenum" suggested by Finney, and widely employed by Kocher, by separation of a similar fascia of adhesion; but it is not generally realized that a similar bloodless mobilization of the ascending and descending colon is possible. Hartmann has employed it with satisfaction on several occasions.

The treatment of tumors of the colon is discussed in a masterly manner by Hartmann. It makes the most interesting reading of any article in the volume. He rightly condemns colocolostomies. For ileocolostomy he prefers the suture to the button. But he always uses the button in the operation of ileorectostomy, employing a special forceps (Lardennois) to introduce the male segment of the button into the rectum (this being done by an assistant), while the female segment is placed in the ileum. The rectum is then incised from its abdominal surface, upon the button, and this, still held in the forceps, is pushed home into the female segment. However convenient the special forceps may be, they are by no means necessary, as a similar operation has been done by Deaver, and possibly by others, without any special appliance. Hartmann's practice of occluding the lumen of the button by cacao-butter, although not new, is a valuable addition to aseptic technique; the butter effectually prevents escape of intestinal contents during the manipulations, and is melted by the body heat in a few minutes after the button is placed.

Hartmann subscribes to Kocher's dictum that it is better to cut arteries and veins, and even to divide a muscle transversely, than to sever a nerve. He thinks nothing of dividing the rectus transversely after a median or paramedian incision, if more room is required to complete the intra-abdominal manipulations. He quotes Wolfler's remark that the gravity of a resection is inversely proportional to the length of the mesentery or mesocolon. If the intestinal lumen is not to be re-established at once he sutures the two loops together "en canon de fusil," and drains the upper by a Paul's tube. He prefers excision and anastomosis for the cure of artificial anus, rather than the destruction of the spur by other means. He prefers lateral to end-to-end anastomosis; and for operations on the large bowel we heartily agree with him. It is better, he says, to make the anastomosis in one of the longitudinal bands.

The statistics given are very interesting, but of course do not lend themselves readily to discussion in a review such as this. It is a pity that throughout the work references are given only in foot notes, and never repeated, further references to a previously quoted author being given as "loc. cit." This slovenly plan gives the reader much unnecessary and uncongenial labor in looking up the authority of a statement. The French also do not understand the value of an index; a table of contents is appended, but it is only because the work is fairly systematic in its treatment of the subjects discussed, that it will be at all available as a book of reference. Apart from these shortcomings it more than sustains the high standard set by the two previous volumes of the same series, and will bring its eminent author added authority and prestige. A. P. C. A.

GENITO-URINARY DISEASES AND SYPHILIS. By HENRY H. MORTON, M.D., Clinical Professor of Genito-urinary Diseases in the Long Island College Hospital. Second edition. Pp. 500 with 158 half-tones and photo-engravings and 7 full-page colored plates. Philadelphia: F. A. Davis Co., 1906.

OF late years the subject of surgical operations for prostatic hypertrophy, inflammatory diseases of the prostate, and the surgery of the kidney have all received particular investigation and study, so that in new treatises on genito-urinary diseases one naturally turns to these topics. The author gives a full chapter on diseases of the prostate, including the various operations upon the hypertrophied gland, and takes the conservative position that it is better to allow the patient to use the catheter as long as he is made comfortable by it, the removal of the gland being postponed until such time as the catheter fails to relieve. Concerning tuberculosis, opinion has changed since the views expressed by Sir Henry Thompson not long ago, namely, that tuberculosis of the prostate is never primary, but always secondary to deposits elsewhere. When the disease is limited to the prostate the surgery of today favors removing all the diseased tissue with a curette, but when the prostate is secondarily involved the indication for operation is not so clear, although even in these cases the subject may thus be saved much misery and suffering, from suppuration and fistula. No chapter in the volume is more interesting than that devoted to operations upon the kidney. On the topic of renal decapsulation in Bright's disease the author sums up the notable work that has recently been done by Edebohls and others, and takes the position that the operation is yet much too recent to express a positive opinion upon its value, time alone being able to show whether another means of prolonging life by surgical operation has been found in a condition hitherto regarded as hopeless.

Syphilis occupies relatively by far the smaller portion of the book. Not much is said concerning Spirochæta pallida, the author maintaining that while it is not yet absolutely proved to be the cause, there is a strong probability that it is the specific organism of syphilis. On the treatment of syphilis emphatic views are expressed in favor of mercurial inunction, as being the most sure and effective mode of treating an ordinary case, and the only plan to be entertained in the presence of grave lesions threatening life or the integrity of vital organs. It is stated that it is possible to give three times the quantity of mercury in this way which could be given by the mouth. Blue ointment (unguentum hydrargyri), made after the formula of the German Pharmacopœia, which contains twenty grains of metallic mercury in every drachm of lard, is the best preparation to use. The author directs attention to the observation that in the United States too much reliance is placed upon the treatment of syphilis by the exclusive use of internal medication, and holds the opinion (and we think justly) that many cases of late tertiary lesions of the brain or nervous system would not have occurred if the patient had been subjected to several active courses of treatment by inunctions or injections during the first two or three years of the disease.

The illustrations are for the most part of a practical kind, some in black and white or merely in outline, some in photographs from life, others in color. The latter are less satisfactory than the former, the process of reproduction employed not being of the highest type of art. The subject, however, is difficult to illustrate. The type is good and clear, the paper heavy and highly calendered. The subject-matter is amply spread out upon the page, with headings and display type, so that the reader has no difficulty in readily finding the topic sought. This feature should attract the student. The views given throughout, on the many topics discussed, are conservative and up to the knowledge of the present period, and they indicate that the author is in touch, from experience as well as from study, with all of importance that has lately occurred in this branch of surgery.

L. A. D.

LECTURES UPON THE PRINCIPLES OF SURGERY. By CHAS. B. NANCREDE, A.M., M.D., LL.D., Professor of Surgery and of Clinical Surgery in the University of Michigan. With an Appendix containing a *resume* of the principal views held concerning Inflammation, by WM. A. SPUTZLEY, A.B., M.D., Late Senior Assistant in Surgery, University of Michigan. Second edition, thoroughly revised. Philadelphia and London: W. B. Saunders & Co., 1905.

THIS volume is composed of a series of lectures on the principles of surgery delivered by the author at the University of Michigan. It is a second edition, the first having appeared in 1899. It is especially prepared for the student and is written in a very comprehensive and systematic manner. We are glad to find the author describing inflammation as a condition entirely the result of microbic infection, and differentiating it in this way from the hyperemia which occurs from simple injury without infection. His whole discussion of this subject is scholarly and convincing. In discussing hydrophobia he has very wisely described the symptoms of this condition in animals as well as in human beings. We are not prepared, however, to agree with his treatment of dog-bites, namely, that of cauterization. This may be desirable if done at once, and if it is known that the dog is suffering from rabies; it is seldom possible, however, to see the patient soon enough after the bite for the cautery to arrest absorption, and it is very difficult to discover whether or not the dog is suffering from hydrophobia. The disease is so rare in human subjects that the most of us could hardly be induced to cauterize with the actual cautery every dog-bite. The omission of chloride of ethyl as a general anesthetic in the chapters on anesthesia is to be very much regretted, as this remedy has been extensively used during recent years. On the whole we can recommend this book as setting forth the principles of surgery thoroughly and in a manner which makes comprehension easy on the part of the student. J. H. G.

Polypus of the Nose. By Eugene S. Yonge, M.D. (Edin.), Honorary Assistant Physician, Manchester Hospital for Consumption and Diseases of the Throat. Pp. 174. London: Sherratt & Hughes, 1906.

Dr. Yonge has written a most interesting monograph upon a subject of vital importance to every rhinologist. The book shows a great deal of original work and very thorough study of the literature of the subject. The general conclusions are in accord with those of most rhinologists. As to the moot subject of etiology, the author enunciates a theory that polyps are œdematous hypertrophies of the nasal mucous membrane due to certain mechanical changes in the glands. To prove this theory he has conducted a series of experiments on the lower animals which he details at length in his book. Although he deserves great credit for the work which he has done, yet it hardly seems that the facts bear out all his conclusions. We believe that the vast majority of clinicians will still continue to believe that, in the larger number of cases, polyps are associated with necrosis of bone or sinus disease. F. R. P.

Diseases of the Nose and Throat. By J. B. Ferguson, M.D., of the New York Post-graduate Medical School. 243 pages with 114 engravings. Lea's Series of Medical Epitomes. Edited by Victor C. Pedersen, M.D., New York. Philadelphia and New York: Lea Brothers & Co., 1907.

This little book thoroughly fulfils the purpose for which it was written, namely, as an outline guide to the subject of rhinology and laryngology. Within a limited space the author has succeeded in covering considerable ground. He has wisely avoided the discussion of theoretical questions, and has dealt with his subject purely from the practical side. For students and general practitioners the book will prove of great use as a brief reference work. It contains a large number of therapeutic formulæ, most of which are of approved value. There are a number of minor typographical errors, especially in the spelling of proper names; thus, Ingals is spelt Inguls, and Ballenger, Bellenger throughout.

F. R. P.

A Study in Nursing. By Miss A. L. Pringle, formerly Matron of St. Thomas' Hospital. London and New York: The Macmillan Co., 1905.

This little book is interesting and useful as an outline of the field of nursing by a nurse of large experience.

The author herself speaks of the work as a "retrospect of an old worker" and very properly states in her preface that the book does not deal with the details of nursing. It does, however, give views of the duties of the probationer, the ward nurse, head nurse, chief nurse, and domestic staff of a hospital, and furnishes an interesting comparative study. There are two points of special interest set forth by Miss Pringle which are at present being much discussed by the heads of training schools, viz., the question of the age of the applicants for admission to training schools for nurses, and the question of the theoretical education of the nurse before entering on the practical field of work in the hospital. There is much to be said in its favor of theoretical studies preceding practical work in the wards; the nurse who had studied thoroughly the necessary theoretical part of the education would be saved the fatigue of attendance on lectures, the study and strain of examinations, and would be of much greater value as a helper in the ward from the beginning. The greater publicity this plan be given the sooner will it meet with deserved success.

J. N. H.

PATHOLOGY: GENERAL AND SPECIAL. A MANUAL FOR STUDENTS AND PRACTITIONERS. By JOHN STENHOUSE, M.A., B.Sc. (Edin.), M.B. (Tor.), Formerly Demonstrator of Pathology, University of Toronto, Toronto, Canada, and JOHN FERGUSON, M.A., M.D. (Tor.), Senior Physician, Western Hospital, Formerly Senior Demonstrator of Anatomy, University of Toronto, Toronto, Canada. The Medical Epitome Series, Edited by VICTOR COX PEDERSEN, A.M., M.D., Lecturer in Surgery, New York Polyclinic Medical School and Hospital. Pp. 285 with 16 engravings and 1 colored plate. Philadelphia and New York: Lea Brothers & Co., 1907.

THE forelying manual contains within a short compass all of general and special pathology that can be crowded therein—which as presented by Drs. Stenhouse and Ferguson is considerable. The arrangement of the subject matter conforms to modern practice, so that the little manual may be used in conjunction with the larger and more detailed books on pathology. The epitomization has been well done and the statements as presented are accurate, so that with the limitations inseparable from such a small book on such a large subject it may be recommended to those desirous of informing themselves hastily on subjects of which it treats.

A. K.

A TEXT-BOOK ON THE PRACTICE OF GYNECOLOGY. By WILLIAM EASTERLY ASHTON, M.D., LL.D., Professor of Gynecology in the Medico-Chirurgical College of Philadelphia. Third edition. Pp. 1097 with 1057 illustrations. Philadelphia and London: W. B. Saunders & Co., 1906.

A THIRD edition of this book is presented to the profession at the end of a year from the original publication—surely an evidence of merit. Changes aiming toward improvement have been made throughout. These include especially the addition of the metric system in the text and formulæ; changes and additions to the illustrations; revision of the chapter on the blood in relation to surgery and special attention to the diagnostic value of polynuclear leukocytosis; the description of a new method of flushing the colon, especially in chronic constipation; amplification of the treatment of vaginismus; and various additions to the operative treatment of cystocele, chronic retrodisplacement of the uterus, intestinal anastomosis, etc. A. K.

PROGRESS

OF

MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF
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A Contribution to Orthostatic Albuminuria.—HEUBNER (*Berl. klin. Woch.*, 1907, liv, 1). The group of cases first classed by Sterling as postural have gradually become separated from the general class of physiological albuminurias as a distinct type. The clinical picture is strikingly uniform, and the recent work of Edel and of Erlanger and Hooker and others has shown a marked relation to blood-pressure changes. Their work suggests that the condition depends upon circulatory derangements and not upon kidney disease. Many prominent authors, however, look with suspicion upon these views and refuse to believe that a normal kidney can secrete such large amounts of albumin over such a long period of time. The opportunity for pathological study seldom occurs, but its importance is evident. Such an exceptional chance has come to Heubner. The patient, a girl aged twelve years, died of a tumor of the brain. She had been under observation at varying intervals for over two years, and during the last year and a half had displayed a typical orthostatic albuminuria. At autopsy the kidneys were found, upon careful microscopic and macroscopic examination, to show only such slight lesions that they could not be held accountable for the symptoms; they were found, indeed, to be practically normal. It is definitely proved, then, that the urine over a long period may contain large amounts of albumin with perfectly healthy kidneys, and such a statement will probably hold good for the whole class of orthostatic albuminurias.

Ileocecal Tuberculosis.—TOMITA (*Wien. klin. Woch.*, 1906, xix, 1581) reports 5 interesting cases of ileocecal tuberculosis. The patients ranged in age from twenty-one to thirty-two years. Two gave a family history of tuberculosis. In all the disease developed gradually with symptoms of intestinal derangement. In two the symptoms followed trauma. The stools were loose or constipated, the two conditions often varying, and after a long period the picture of a chronic obstruction developed, with a palpable tumor in the iliac fossa, in at least one instance discovered by the patient himself. The tumors were about the size of a fist, elastic, slightly tender, and somewhat movable. In two cases there were enlarged tuberculous mesenteric glands as well as the tumor. All of the patients did well after operation excepting one, who died as the result of necrosis, probably from the pressure of a Murphy button. Even the patient from whom the mass could not be entirely removed on account of adhesions was greatly improved. Of special interest clinically is the statement that no tuberculous lesion could be found in any other part of the body. Since the time of Billroth these ileocecal tuberculous tumors have been grouped as a special clinical entity, and there is a large mass of literature contributed to the subject. They simulate very closely the scar-tissue masses following appendicitis and cecal carcinoma, and mistakes in diagnosis are frequent. Operation with extirpation of the mass is followed by good results. In 85 cases collected by Conrath there were 11 deaths.

Blood-pressure Estimations in Man.—Bing (*Berl. klin. Woch.*, 1906, liii, 1650) states that the ultimate aim of all blood-pressure estimations is to obtain an index of the pressure in the aorta. Experimentally this is done by inserting a cannula into the large branches of the aorta and measuring the end pressure. Bing directs his investigations toward discovering if the measurements obtained by the usual methods are the terms of end or lateral pressures. If we obtain lateral pressures these will depend not only upon the pressure in the aorta, but upon the degree of contraction of the vessels distal to the point of compression. A sudden dilatation of the small arteries of the arm would, if the pressure were read from the brachial, show a fall which would by no means indicate a parallel fall in the aorta. From his experiments, which consist in measuring the pressures in both brachials and by various means causing the arteries in one arm to dilate or contract, and then noting the variations in pressure in the two brachials, Bing concludes that what we obtain are lateral and not end pressures. This fact should be borne in mind when we draw clinical deductions, particularly deductions from the effects of therapeutic measures.

Pseudochylous Exudates.—Zypkin (*Wien. klin. Woch.*, 1906, xix, 1032). Exudates with a milky appearance have been divided into three classes: the hydrops chylosis, the hydrops chyliformis or adiposis, and the hydrops pseudochylosis. In the first group are the fatty exudates containing chyle from the lymphatics. In the second fat is likewise present, but according to Quincke it is derived from degenerated epithelial cells and is not due to the presence of chyle. In some instances no doubt the two conditions are united. In order to obtain an opalescent emulsion 1.5 per cent. of fat must be present, and any milky exudate with less fat must depend for its color upon some other substance. In cases of hydrops adiposis the fat alone is not always responsible for the color, as the hydrops often retains a milky appearance after all fats have been removed.

Numerous substances have been isolated and considered the cause of this color. In the two instances he reports Zypkin obtained lecithin, globulin, nucleo-albumin, and a mucoid substance, but he does not make any of these responsible for the opalescent appearance, as the same substances were obtained after the exudate had become clear, and, indeed, were found in the pleural transudate of a heart case with broken compensation. Pseudochylous exudates are not distinguishable chemically from simple serous exudates, the milky color depending probably upon some change in the solubility of one of the constituents, perhaps of one of the four substances isolated. It was observed that as the exudate lost its milky appearance the albumin content increased. Achard has noted that in some conditions the blood may become so poor in albumin that the serum assumes a milky color. Zypkin applies that observation to pseudochylous exudates, and suggests that the solubility in whatever the substance is, which causes the milkiness, depends upon the percentage of albumin present.

Mixed Infection in Pulmonary Tuberculosis and its Relation and that of Intestinal Tuberculosis to Amyloid Degeneration.—The frequency and importance of mixed infection in pulmonary tuberculosis has never been settled definitely. The determination of its presence is not by any means easy; the foreign organisms obtained may not come from the disease focus, and even if they do, they may have been there as harmless parasites. To prove that the microörganisms discovered in the examination really come from the diseased lung focus, it is necessary to wash thoroughly, after one of the approved methods, the sputum particles, to be examined either by stain or culture. Organisms having attached themselves in the bronchial passages or elsewhere are thus easily removed. For culture purposes media must be employed suitable both for tubercle bacilli and other organisms; egg albumen as a culture medium is probably the best. Another unsettled point is the question of the significance of organisms other than the tubercle bacilli in the production of symptoms usually ascribed to mixed infection; many observers insist that all the symptoms and lesions discovered can be produced by the tubercle bacillus without assistance; this refers in particular to bronchitis, pleurisy, bronchiectasis, pneumonia, hectic intermittent fever, etc. J. Sorgo's observations (*Ztschr. f. klin. Med.*, 1907, lxi, 250) on these points gave the following results: The sputum of 30 patients, 28 of whom were in the third stage of tuberculosis, was examined; 18 had remittent, 3 intermittent, and 9 no fever; 1 patient had amyloidosis. Tubercle bacilli in pure culture were found in the sputum of 26 (among them the hectic fever and amyloid patients); 4 had mixed infection. Of 19 individuals who came to autopsy tubercle bacilli alone were found in 13, mixed infection in 6. In 5 pleurisy cases the serous exudate and sputum contained tubercle bacilli only, in 2 staphylococci were also found, but in 1 they migrated probably after death. Of 17 cadavers examined bacteriologically only 3 had a mixed infection; in 3 mixed infection was doubtful, while in 11 only tubercle bacilli could be found; all types of lesions contained nothing but tubercle bacilli. Sorgo concludes that all the clinical symptoms and all the pathological lesions can be induced by the tubercle bacillus without the aid of other organisms; mixed infection, while it occurs, is not nearly of the importance which has been ascribed to it. Of 9 cases of general amyloid disease which he has studied 8 had tuberculosis of the intestines; 5 marked cases had deep intestinal ulcers, 3 cases with microscopic lesions only had but slight ulceration. Pure cultures of tubercle bacilli were found in 6, mixed infection in 2. The ninth case had no intestinal complication, but had marked mixed infection, with staphylococci and streptococci. Sorgo believes that amyloid degeneration in tuberculous patients means either mixed infection from the standpoint of the lungs or intestinal tuberculosis. The presence of a pure culture of tubercle bacilli in the sputum with positive amyloid degeneration points to the existence of an intestinal tuberculosis. One such clinical case was observed.

The Relationship between Simple Posterior Basic Meningitis and Cerebrospinal Fever.—F. Langmead (*Practitioner*, 1907, lxxviii, 485) is of the opinion that posterior basic meningitis is sporadic cerebrospinal fever, modified, but not differing from it any more than one epidemic differs from another. He discusses the salient clinical and pathological features of posterior basic meningitis as it is met with in infants and the symptoms of the disease as it occurs in older children, and he tries to show that the latter forms a link between the disease as it is found in infants and cerebrospinal fever. After discussing the symptoms, complications, pathology, and bacteriology of posterior basic meningitis he notes the similarities between the two diseases. In both there are initial coryza, irritability, and headache; in both there are rigidity, head retraction, strabismus, and spasms; and in both hydrocephalus is the most common sequel. The chief differences are: The rarity of the rashes in infants with meningitis—older children have them in 20 per cent., in cerebrospinal fever they occur in 33 per cent. of the cases; optic neuritis occurs in 7 per cent. of infants with posterior basic meningitis, in 50 per cent. of older children with posterior basic meningitis, and in 15 per cent. of patients with cerebrospinal fever; amaurosis occurs in 33 per cent. of infants with posterior basic meningitis, in 10 per cent. of older children with posterior basic meningitis, and rarely in cerebrospinal fever; the exudate in posterior basic meningitis is at the posterior base and about the cord, in cerebrospinal fever at the vertex, base, and cord alike. The diplococcus of Still, the cause of posterior basic meningitis, differs from the bacillus of Weichselbaum, the cause of cerebrospinal fever, only in its readier growth and greater vitality; but recent experiments are nullifying these distinctions. Still's bacillus has been isolated from the blood of a case of malignant epidemic cerebrospinal fever. Epidemics of posterior basic meningitis have been noted.

Mechanical Stimulation of the Secretion of Gastric Juice.—The statement accepted for many years, that the secretion of gastric juice is incited as much by mechanical as by chemical irritations has been contradicted by Pawlow and his disciples, who claim that gastric juice is secreted partly as the result of the psychic influences induced by the appetite and partly by chemical influences induced by the food itself. Their statement that the mechanical properties of the food substances are not capable of producing gastric juice, is based upon the fact that they irritated the gastric mucosa by repeated inflation with a rubber ball, with feathers, and a glass rod, and by blowing coarse sand into the stomach. A. Schiff (*Ztschr. f. klin. Med.*, 1907, lxi, 220) comes to a different conclusion. He fed hungry dogs with milk and with milk and bismuth subnitrate. The first dogs secreted 5 and 7.5 c.c. in one and one and a quarter hours, respectively; the second 8.5 and 14 c.c.. The same results were noted when fine sand and a mixture of the two with bismuth were used. Experiments, in which fine sand and glass powder were introduced into the empty stomachs of dogs, who had been watched for an hour, produced similar results. Schiff explains the differences between his and Pawlow's results by stating that the method which Pawlow employed does not resemble the physiological conditions in the slightest. He believes that his experiments justify the conclusion that the mechanical irritation and the mechanical weight of the ingesta have a proportionate influence on the amount of gastric secretion. He concedes that the mechanical secretion is not as great as that of the chemical irritation of ingesta and that both vary in different animals.

Free Pulmonary Hemorrhage in Mitral Stenosis and Extensive Sclerosis of the Pulmonary Arteries.—G. Schwarz (*Münch. med. Woch.*, 1907, liv, 615) reports the case of a man, aged thirty years, who began to complain of palpitation and dyspnœa at the age of twenty-four years. He had several attacks of hemoptysis at twenty-eight, and at that time complained of a sensation as if something had torn in his heart. Since then he has had several hemorrhages of variable amounts, and has frequently expectorated blood during prolonged periods. Examinations two weeks before death showed marked anemia, some rales in the lower right lobe posteriorly, the heart enlarged in every direction, the apex-beat in the sixth interspace, a presystolic murmur at the apex, a systolic murmur at the pulmonary orifice with accentuation of the second sound, roughening of the first sound over the tricuspid area, pulsating cervical veins, and albuminuria; the sputum was bloody, contained cardiac cells but no tubercle bacilli. During the next two weeks the patient expectorated about 100 c.c. of blood daily, and two days before death developed a right-sided paralysis. Death was preceded by a large hemorrhage, dyspnœa, and cyanosis. The autopsy revealed an embolus in the left Sylvian artery, the cause of the paralytic attack, mitral and tricuspid stenosis, brown induration of the lungs, hemorrhage in the bronchi, advanced sclerosis of the pulmonary arteries, and infarcts in the spleen and the kidneys. Attention is called to the profuse pulmonary hemorrhage, which had its origin in the marked congestion due to the mitral disease and not in tuberculosis. Cases of this kind are usually spoken of as cardiac hemoptysis. Sclerosis of the pulmonary arteries in most instances does not betray its presence. Bleeding, as in the case reported, may be due to embolus of the pulmonary arteries, pulmonary congestion, degeneration of vessel walls, etc. In this case the vessels showed no rupture anywhere, but rather the picture of endarteritis proliferans, and the hemorrhage must be ascribed, therefore, absolutely to the congestion.

Venous Murmurs in Cirrhosis of the Liver.—Investigations by G. CATTI (*Ztschr. f. klin. Med.*, 1907, lxi, 269) have shown that venous murmurs adible over and in the neighborhood of the spleen are relatively common in cirrhosis of the liver, occurring in one-fifth to one-sixth of all the cases; murmurs audible elsewhere, as in the epigastrium, near the xiphoid, above the umbilicus, etc., are very uncommon and are still unexplained. His first patient had hepatic cirrhosis to a marked degree; the veins about the umbilicus were somewhat dilated, those of the rest of the abdomen very markedly. The xiphoid, mammary, and inferior epigastric veins were markedly dilated and connected by a number of branches. Several centimeters to the left of the umbilicus the hand could feel an intense thrill, most marked over the left inferior epigastric vein; it was easily heard over the entire abdomen, but varied in intensity and character in different places. Compression of the left epigastric vein below the point of greatest intensity caused a disappearance of the murmur; below this point the vein was narrower than farther up. The murmur was evidently caused by the blood flowing suddenly from the relatively narrow inferior epigastric vein into the more dilated superior epigastric vein; the junction of the deep and superficial inferior epigastric was immediately below this point, increasing the pressure, and the blood pressure was still farther increased by the stasis in the portal vein. In the case of his second patient a venous murmur was audible but not palpable immediately below the ensiform; it was transmitted upward along the sternum. The cause of this he found in the junction of the para-umbilical veins, or probably of the termination of the umbilical vein in the larger xiphoid vein, or finally of the deep left epigastric vein into the para-umbilical veins. These murmurs, therefore, are due to the blood flowing under considerable pressure from a relatively narrow vein into a larger one, in which the pressure is considerably lower; they are murmurs of stenosis.

SURGERY.

UNDER THE CHARGE OF

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The Surgical Treatment of Prostatic Hypertrophy.—LEGUEU (*Annal. de la policlin. cent. de Bruxelles*, 1907, vii, 19) says that perineal prostatectomy is less grave, but does not always cure; while the suprapubic operation is more grave, but cures completely when it does not prove fatal. Legueu prefers the latter, and considers it the operation of choice in the greater number of cases that are suitable for a prostatectomy. He considers, however, that the perineal operation should not be abandoned, but should be reserved for many cases in which it would be rash to attempt the high operation. The indications for the two operations are considered from the standpoint of the prostate and from that of the patient.

On the part of the prostate the volume is most important. The larger the prostate and the greater the prominence in the bladder the stronger the indication for the high operation. The removal of a large gland is always difficult by the low operation, and it is often impossible to make it complete. The high operation is also indicated whenever there is an important median lobe. One should reserve for this operation those prostates which are frankly enucleable, that is to say, the adenomatous, and should leave for the perineal operation those with diffuse enlargement, indefinite outline, and firm consistency. By the perineal route piecemeal removal is more effective than by the suprapubic.

On the part of the patient the contra-indications are concerned with constitutional conditions lowering the resistance of the patient. The suprapubic operation, constituting a grave trauma, should be reserved for patients in good condition, those who are young in spite of their age, without marked obesity or atheroma. Those with opposite conditions are more liable to emphysema and pulmonary congestion than with the perineal operation. In infected patients who have or have had fever, the perineal operation will give dependent drainage, while the suprapubic operation only increases the dangers. If the genital functions are to be preserved the high operation should be done. The latter, therefore, should be performed, except in those patients with very small prostates, in fat subjects, and those with infections.

Comparative Results of Perineal and Suprapubic Prostatectomy.—Pousson (*Annal. des maladies des organes genito-urinaires*, 1907, xxv, 241), after having been a warm partisan of the perineal operation, justifies his change to the suprapubic in an analysis of 50 personal total prostatectomies, by the two routes, 28 perineal and 22 suprapubic. The mortality of the first group was 4 deaths (14.3 per cent.) and of the second 5 deaths (22.7 per cent.).

Although the advantage as to mortality was slightly in favor of the perineal operation, Pousson argues for the suprapubic for several reasons. The anatomy of the latter is the more simple, the only obstacle being the peritoneal cul-de-sac, which is easily avoided. By the perineal route the bulb of the urethra and the rectum are the structures most difficult to avoid. That wound of the bulb may be grave Pousson has had the opportunity of proving in several cases. Opening the rectum is more grave, and one is never sure that it will not occur at the time of the operation or later from the separation of a sphacelus. Pousson, in his 28 perineal prostatectomies, perforated the rectum twice during the operation, while in a third case perforation occurred secondarily on the tenth day from mortification of the tissues. The last closed spontaneously, but the first two gave rise to permanent fistulæ.

The enucleation by the suprapubic route has always been easy and rapid, while by the perineal it has often been tedious and laborious. It is particularly in what Pousson calls the pelvic form of the enlargement that the perineal operation is difficult. In this form the posterior superior angles of the gland push backward, forming two prolongations on the sides of the pelvic cavity. It was frequently necessary to remove the prostate piecemeal by the perineal operation, while by the suprapubic he was always able to remove it in two fragments or as a whole.

The most frequent obstacle in the perineal operation was excess of fat. In such cases the suprapubic route permits a larger incision, which facilitates the enucleation and allows the introduction of the whole hand into the bladder. The excessive hemorrhage which many surgeons fear in the suprapubic operation Pousson has found in only two cases. Both required a tampon of the bladder, after the patient had been put back to bed, but both recovered. While the drainage is better after the perineal operation, he has had very little trouble on this account in the suprapubic. The advantage from the standpoint of sepsis is in favor of the suprapubic operation. The average time for the healing of the wound was 5.5 weeks in the perineal operation and 4.2 in the suprapubic. Pousson makes a large opening in the bladder, and aims to expose the field of operation to the eye by means of retractors and an electric lamp. With a metallic hood having a lateral opening to which is fitted a rubber tube, he has been able in several cases to drain through the suprapubic opening for five or six days without a change in or saturation of the dressing by urine. At the end of that time he drains by a catheter in the urethra.

A Contribution to the Anatomical and Clinical Study of Cancers of the Prostate.—Motz and Majewski (*Annal. des maladies des organes genito-urinaires,* 1907, xxv, 161) base their study upon 60 cases, of which 26 were diagnosticated clinically, and 30 only after autopsy. Of the 26 diagnosticated clinically, a considerable increase in volume was rare (2 cases), a moderate increase occurred in the great majority (17 cases), in 4 the gland was of normal size, and in 3 it appeared to be smaller than normal. The size has no relation to the gravity. Some small prostates showed considerable invasion of the corresponding glands and often enormous tumor masses along the vertebral column. The diagnosis depends chiefly upon the irregularities and the hardness. Variations, however, in the consistency are frequent. In some cases in which a vesical tumor was recognized, it was found to be only a propagation of a prostatic tumor which was not recognized. The urethra was invaded in only 7 cases (27 per cent.). In only 1 case was involvement of the bulb and of Cowper's gland observed. Of 38 cases of tumor of the bladder one of the writers has found that two-thirds (28) were originally prostatic tumors, and this frequency of propagation from the prostate to the bladder is generally admitted. Of the writers' 26 cases diagnosticated clinically, in 20 the seminal vesicles were involved. The lymphatic glands were invaded in nearly all cases, only 1 showing a negative result from this standpoint. Not a single case was found to have invaded the rectum, and only 3 could be found in the literature. This speaks against the existence of the prostatorectal lymphatics claimed by some authors. In 3 cases the peritoneum was invaded. As to metastatic deposits the observations of the writers are not precise. They found mentioned the following cases: 3 to the liver, 2 to the pleura and lung, 1 to the kidney, and 1 to the femur. They found 2 cases, not included in the 26, in which vesical cancers invaded the prostate.

Of the writers' 60 patients, in the great majority the first symptoms were those of prostatism, 8 presented complete retention as the initial symptom, and 38 began with the ordinary dysuria of prostatics, and incomplete evacuation of the bladder. In 5 cases neuralgia appeared as the first symptom; in a few hematuria was the initial symptom. In other cases these symptoms developed later, and were usually due to an associated periprostatitis. Neuralgias referred to surrounding regions, as lumbago, sciatica, etc., were usually due to the pressure of propagated masses upon surrounding nerves. The writers conclude that hematuria results from the propagation of the cancer to the bladder or posterior urethra. In 40 per cent. of their cases the duration between the first appearance of symptoms and the fatal termination was not more than six months. Early diagnosis was impossible in at least half of the cases. Often the cancerous process is far advanced at the time of the appearance of the first symptoms. There are, however, some cases in which rectal palpation gives the impression that the prostate alone is involved, and it is only in these that extirpation should be tried, even then only after endoscopic examination of the posterior urethra and bladder. Of 5 cases reported in which recurrence had not taken place up to the time of publication only 2 were sufficiently followed to be at all reliable, 1 of them for nine months and 1 for eight months. Up to the present they have not had a single case of definite cure.

The Technique of Exposure and Excision of the Cervical Sympathetic.—Sebileau and Schwartz (*Rev. de chir.*, 1907, xxvii, 161) make their incision along the anterior border of the sternomastoid muscle, from the mastoid process to the level of the hyoid bone. The incision should be made rather a little back of the anterior border of the muscle in order not to damage the posterior lobules of the parotid gland. When the sheath is divided the anterior edge is grasped by forceps and carefully dissected from the muscle anteriorly and from the deep surface of the muscle. The sternomastoid is then strongly retracted posteriorly, making the spinal accessory nerve which lies under the sheath tense and slightly prominent The nerve is then liberated without difficulty and is in no further danger. Moreover, the sheath of the parotid is unopened and the gland need not be seen. One can expose the sympathetic either below or above the spinal accessory. The latter route is the more difficult, although one cannot expose fully the superior cervical ganglion. In the high exposure the sternomastoid is more adherent to its thickened sheath, the connective tissue is more dense and less supple, the bloodvessels are exposed and retracted with greater difficulty, and finally one must dissect close to the transverse process of the atlas in the obscure depths of the vertebropharyngeal space. The lower operation, therefore, is better.

The deep layer of the sheath of the sternomastoid may be divided on a grooved director posterior to the bloodvessels which are masked by it. This is done externally to the line of the anterior tubercles of the transverse processes of the vertebræ, on a grooved director. This is aided by the marked prominence in the depth of the upper part of the wound of the first transverse process. The vascular nervous sheath is then freed by the use of a grooved director. Only the vein should be caught by the retractor, lest the sympathetic be masked by the other contents of the vascular sheath, to which it is adherent by its own thin sheath as it lies underneath. The sympathetic nerve is attached to the prevertebral muscles under the carotid. The nerve with its ganglion is sometimes replaced by a plexiform arrangement. One sees coming from it internally and below an important branch, the superior cardiac nerve. In order to disengage the head of the superior ganglion, it is necessary to go deeply into the cavity under the parotid gland.

The Operative Treatment of Wryneck.—GERDES (*Zentralbl. f. Chir.*, 1907, xxxiv, 145) says that while the total or partial extirpation of the contracted sternomastoid, the operation of Mikulicz, has produced good results in the hands of some surgeons, recurrences have occurred in the hands of others. Trendelenburg emphasized the defective cosmetic effect and recommended, in addition to the tenotomy of the sternomastoid, the division of the cervical fascia. Wullstein proposed, besides extirpating the affected muscle, to shorten the stretched one of the opposite side. Other muscles than the sternomastoid are involved, as the scaleni, levator anguli scapuli, and the acromial portion of the trapezius. Next to the sternomastoid the anterior scalene is most frequently involved. Gerdes describes his method of dividing the sternomastoid and the anterior scalene muscles. A transverse incision 5 to 6 cm. long is made a finger's breadth above the clavicle, beginning over the sternal portion of the sternomastoid. After dividing both origins of the muscle, the cervical fascia posterior to it is opened freely and the external jugular vein exposed. The omohyoid muscle is retracted upward and outward and the external jugular vein inward. The anterior scalene muscle is then exposed with the phrenic nerve lying on it and the brachial plexus external to it. After careful isolation of the muscle from these structures it is divided on an elevator or similar instrument just above the subclavian artery, which it covers. If necessary, by enlarging the wound, the scalenus medius may also be divided. The small wound is packed with gauze for forty-eight hours and is then permitted to heal by granulation. In about four days passive and active movements are begun and are continued for three to six weeks. The cosmetic and functional effects are satisfactory even in old cases, so that in Gerdes' judgment this is a better operation than that of Mikulicz.

The Pathology and Treatment of Neuralgia, Particularly by Neurosarkokleisis.—BARDENHEUER (*Jour. de chir. et annal. de la Soc. Belge de chir.*, 1907, vii, 72) says that exceptionally medical treatment is followed by a cure, while the different operations, except the removal of the Gasserian ganglion, give only about 20 per cent. of cures and 80 per cent. of recurrences. The affection has been ascribed to various causes, as tumor formation in the interior of the nerve, to changes in the internal coats of the arteries of the nerve, to changes in the blood supply of the nerve from vasomotor disturbances, and to disturbances of the venous circulation at the base of the skull. Bardenheuer also believes that the cause lies in disturbances in the circulation in the veins which accompany the nerves through the bony canals by which they leave the skull, the spine, and the bones of the face. In 8 operations for sacro-iliac joint disease he observed that the pain did not result from an extension of the tuberculous process, but simply from a passive, pernicious, venous hyperemia. The cure of the joint disease was simultaneous with the disappearance of the sciatica. In 5 cases of facial neuralgia he found the trunk of the nerve markedly inflamed at its exit from the canal. He explains this condition as follows: In consequence of exposure to cold, trauma, friction, alveolodental periostitis, etc., a peripheral perineuritic hyperemia develops up to the osseous canal where the stasis is arrested by the bone. Repetition of this phenomenon produces an attack of neuralgia from compression in the canal. In time the hyperemia traverses the canal to the roots of the nerve and the ganglion. His arguments are as follows: Each time that he operated he established the existence of the venous hyperemia, the pain disappeared immediately after each operation, the cure lasted six to eight years in all cases, no damage was done to the nerve, and the sensation was intact afterward.

Bardenheuer recommends a new method of treatment which he calls neurosarkokleisis. It consists in removing one of the walls of the bony canal and transposing the nerve to the soft tissues. This resulted in complete success in 8 cases of sciatica and 4 cases of facial neuralgia. A fifth case of the latter condition recurred because the lower jaw was broken, with a wound opening into the mouth. This wound healed by second intention, and, therefore, with many adhesions to the nerve. He attacks the first division of the nerve at the supra-orbital foramen, the second division by removing the roof of the infraorbital canal in the floor of the orbital cavity, and the third division by removing the covering of the whole of the dental canal.

A New Method of Dressing Oblique Fractures of the Leg.—DESGUIN (*Jour. de chir. et annal. de la Soc. Belge de chir.*, 1907, vii, 78) offers a new apparatus for the correction of deformity and the maintenance of reduction in these frequently troublesome fractures. He says, however, that he is a frank partisan of the open operation for these cases and believes that a complete cure, from an anatomical and functional point of view, can be obtained only in this way. Unfortunately operation requires favorable conditions that cannot always be combined in a given case, such as a convenient place, proper assistance, appropriate material, and anesthesia. The greatest difficulty in the non-operative treatment is to obtain suitable reduction of the fragments, and the greatest factor in accomplishing this is extension. Desguin offers an apparatus which maintains reduction, and at the same time permits motion in the knee and ankle, so that the function is little disturbed in these joints as well as in the muscles which will not atrophy. It favors the circulation, thus maintaining the vitality and helping in the union of the broken bones.

The apparatus consists essentially of two inferior plates, which are fixed to the limb below the fracture by a cuff of plaster of Paris. Two superior plates are similarly fixed to the limb above the fracture. Each inferior plate is furnished with a kind of rack fixed transversely and containing several deep notches. Each superior plate carries also a transverse plate, but this is perforated with four or five rectangular openings. These transverse plates, the upper and lower, are held together by two long bolts, with nuts on each side of the leg, fitted into the rectangular openings of the upper plates and the corresponding notches of the lower. The paper is admirably illustrated with fifteen photographic plates. Desguin has employed the apparatus in three very bad cases with very satisfactory results. He does not claim, however, that it makes the treatment of these fractures simple, and believes that an open operation is easier. Any treatment of these fractures is difficult, as is shown by the bad results generally obtained. He has simply tried to improve the non-operative treatment.

THERAPEUTICS.

UNDER THE CHARGE OF

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The Treatment of Hyperchlorhydria.—L. Furst (*Wien. med. Presse*, 1906, No. 20, p. 1091), in this affection advises meals of small bulk and of non-irritating foods; of those allowable, he mentions cocoa, thin and purée soups, lean meat cooked rare, fish (the lean portions only), stewed fruits, and various green vegetables. Condiments, sea food, fat meats, acid salads, and dried fruits are to be forbidden. Potatoes, sweets, and fresh bread in small amounts may be permitted. Sparkling, acidulated waters with a little red wine and light beer may be taken. By diet regulation the excess of hydrochloric acid and the excessive mucus may be diminished, while the dilatation and gastric atony may be benefited. In connection with such a diet as that mentioned Furst advises the administration of alkalies to combat the eructations, nausea, flatulence, etc.; here, if these symptoms are merely transient, sodium bicarbonate is advised, but if continued treatment of these manifestations is necessary he prefers the alkaline mineral waters, such as Vichy. The waters should be taken in sufficient amount, about a half-hour after each meal.

Hepatic Extract in Atrophic Cirrhosis.—J. Carles (*Revue de thérapeutique*, 1906, No. 11, p. 387.) reports the history of a patient suffering from atrophic cirrhosis with ascites. Upon admission to the hospital paracentesis was performed and about seven quarts of fluid was withdrawn. About seven hours later a second tapping seemed necessary, but hepatic extract was prescribed instead. Marked and rapid diuresis resulted and the general state of the patient became much improved. This form of treatment, according to the observers, should give excellent results in the precirrhotic stage of this disease. Other clinicians have employed the hepatic extract without accident and with good effect. One reports a series of cured cases of cirrhosis, and states that those most amenable to this form of treatment are those affected with ascites due to mechanical causes.

The Internal Employment of Adrenalin.—S. KREUZFUCHS (*Wien. med. Presse*, 1906, No. 17, p. 921) states that adrenalin has distinct limitations as an internal medicament, being contraindicated in arterial hypertension and, in all instances, for continued administration. He has employed it in an instance of angioparalytic hemicrania with good results, the headache having ceased within a half-hour and sleep being induced, from which the patient awoke refreshed. In the angiospastic type of the affection the drug is contraindicated and it is useless in simple headaches. In nervous dyspepsia associated with painful paroxysms and nausea a pill containing 1/120 grain caused a quick relief of the symptoms. Adrenalin, while a drug possessing only limited applications, seems to be an efficient remedy in the relief of nausea and vomiting.

The Injection Treatment of Neuralgia.—According to P. KRAUSE (*Allg. med. Central-Zeitung*, 1906, No. 16, p. 291) various agents, such as antipyrine, methylene blue, cocaine, alcohol, ether, osmic acid, etc., have been employed in this connection. He warns against the injection, particularly of the last three, into mixed sensory and motor nerve trunks, on the ground that permanent paralysis may be produced. He has used 0.1 to 0.2 per cent. of stovaine in 8 per cent. saline solution to which 1 to 1000 adrenalin chloride (1 part of the latter to 300 to 600 parts of the stovaine solution) has been added. The adrenalin causes a local anemia which is succeeded by a hyperemia. The treatment has temporarily relieved two instances of sciatica and has effected permanent cure in two patients afflicted with trigeminal neuralgia, the injections having been made at the points of emergence of the nerve. In the cases of sciatica 1500 minims were injected, in the others only 300 minims. The injection, in addition to a local anesthetic action, has a mechanical effect upon the nerve owing to the considerable amount of fluid injected. In the author's hands intraspinal injections of stovaine have relieved the lancinating pains of locomotor ataxia in two instances.

A Case of Veronal Poisoning.—JOHN GERMANN (*Jour. Amer. Med. Assoc.*, 1906, No. 26, p. 1999) reports an instance of fatal poisoning from 200 to 250 grains of veronal. The drug was taken because of the impatience of the patient to get to sleep. When found he could not be roused. The pulse was 90, the respiration 24. A half-hour before death, seventy-nine hours later, the pulse was 152, the respiration 44, the temperature 108.5°. During the last twenty-four hours of life respiration was irregular and stertorous and the patient was cyanosed. The quality of the pulse was good until the last four hours. After twenty-four hours the urine contained albumin and 33 per cent. of this substance was present just before death. The patient remained in a comatose condition until death supervened. Postmortem, the brain was only slightly œdematous, but this with the other organs was markedly congested; the blood was chocolate colored.

The Employment of Physical Agents after Operations for Gallstones.—Schurmayer (*Therapeutische Monatshefte*, 1906, xx, 483) considers that there is much more in the treatment of cholelithiasis than is afforded by surgery. The painful gastric crises which sometimes occur, he treats by early morning lavage with hot water, followed by the introduction into the stomach of a pint of 1 to 1000 silver nitrate solution, which is allowed to remain in the organ for from ½ to 2 minutes; after this solution is withdrawn the viscus is again washed with water. As a substitute for the silver solution he sometimes employs a mixture of a quart of lukewarm water and a half-ounce of chloroform. Intragastric faradism is also employed and by this means the flow of bile is excited; following this measure a diminution in the size of the liver, a disappearance of the pain, and an increased appetite may be observed. As a local application Schurmayer prefers the electric thermophore; in certain instances the electrostatic bath and massage achieve good results. In excessive meteorism the faradic current, one large electrode being applied to the back, the other to the abdomen, will give relief. Constipation, which is a common sequence of operation, should be treated by means of massage and respiratory gymnastics, and for the right nephroptosis, which is not an unusual sequel, the employment of vibration is advised.

Beer Yeast in Infantile Gastro-enteritis.—Sittler (*Münch. med. Woch.*, 1906, No. 36, p. 1761) has used beer yeast in infantile gastro-enteritis in doses of 15 to 45 grains daily of the dry yeast, the amount depending upon the age of the patient. The patient's milk is stopped and sugar solution is substituted, and, if necessary, gastric and intestinal lavage are employed. The yeast has yielded excellent results in cases in which there has been excessive intestinal fermentation; the fœtor of the stools is diminished and their number is lessened as a result of its action. Merely the administration of the yeast suffices to effect a cure in some instances. When it fails calomel and astringents are employed as adjuvants. The treatment never increases the diarrhœa, and it is indicated in severe instances of the disease as well as in those of mild type.

The Treatment of Paroxysmal Tachycardia.—Hay (*Edin. Med. Jour.*, 1906, xxi, 40) states that in order to prevent recurrence of the paroxysms we should endeavor to restore the normal stability of the cardiac mechanism. Breathing exercises should be undertaken and all sources of reflex irritation, such as the kidneys, digestive tract, or uterus, should be treated, if necessary. Tea, coffee, and tobacco should be forbidden; during attacks the patient should be kept in bed and on a light diet. In the presence of nervous irritability the bromides are useful. Attacks sometimes may be cut short by such mechanical methods as holding the elbows tight to the chest, forcibly compressing the abdomen, and holding the breath after a deep inspiration. Pressure upon the vagi may be tried and deep respiratory efforts may cause a cessation of the paroxysm. Of drugs, digitalis, by mouth or hypodermically, is most often prescribed, but the attacks often persist in spite of heroic dosage. It is probable, however, that, even though it may not cut short the paroxysm, the drug is beneficial, since it has been shown to have a specific action on the auriculoventricular bundle. If portal congestion and venous stasis are present they should be relieved by calomel and the salines, and ice over the precordium, mustard poultices, and morphine should be used to mitigate pain and distress. The height of the blood pressure should always be estimated and taken into account, because, as a rule, the arteries are contracted upon their diminished contents and give erroneous impressions to the fingers. If there is increase of blood pressure the hippurates, benzoates, and nitrites will be of value. When contractility is impaired, as shown by the alternating pulse and visceral engorgement, calcium chloride may be given with advantage. In Hay's opinion treatment at present is largely empirical, and our endeavor should be to find a drug or combination of drugs which will depress the function of stimulus production that is possessed by the muscle fibers which join the auricles to the ventricles.

Alcohol and Fats in Diabetes.—ROMME (*La presse méd.*, 1906, No. 89, 712) has come to the conclusion that the acetonuria which occurs in diabetic patients who are subsisting upon a diet of proteid and fat, and who are getting no carbohydrates, is due to the presence of the fatty elements of their regimen. The healthy individual, when put upon a diet of fat and proteid only, exhibits an increased elimination of acetone, and this augmentation is more marked in the diabetic. To obviate the difficulty the author suggests that alcohol be substituted for the fat. The quantity should be about 10 drams daily in the form of wine, beer, tea *au rhum*, etc. The diabetic patient bears the alcohol well and it is likely to cause neither excitation nor depression.

Dechloridation in Nephritis.—M. BOURGET (*Revue médicale de la Suisse Romande*, 1906, No. 4, p. 249) considers this mode of treatment merely a fad in therapeutics and of little value in the diminution of dropsical conditions. According to Bourget œdema may be effectually lessened by the administration of purgatives and cardiac tonics in connection with the potassium salts, the acetate being an especially efficient diuretic. It is easily transformed into potassium carbonate, which is eliminated as the chloride. At the same time dechloridation may be performed by having the patient take his potatoes without salt.

Copper Solutions in Intestinal Amœbiasis.—WILLIAM R. MOULDEN (*The Medical Record*, 1906, No. 1864, p. 132) has employed intestinal instillations of copper sulphate solution of a strength of not over 1 in 6000 at temperatures of 106° to 110° F. in amœbic infections, with excellent results. The instillations were given every twelve hours, the buttocks being raised so as to allow complete irrigation of the colon, especially at the cecum, where the amœbæ are usually present in large numbers. The colon was thoroughly irrigated with sterile water until the return was perfectly clear. After draining off all the surplus water the bowel was slowly filled with the copper solution by starting with the reservoir on a level with the anus and slowly elevating it as the bowel accommodated itself to the pressure, thus distending it to its fullest capacity without exciting contraction of its muscular walls. The solution was retained usually without difficulty for twenty minutes. At 2 o'clock in the morning each patient was given 1½ ounces of saturated magnesium sulphate solution, with the object of washing from the membrane by exosmosis as many of the amœbæ as possible, and, by clearing out the digestive residue, securing a clean tract for the day's work. The results of the copper treatment were compared with those obtained in a similar series of patients treated by quinine irrigations, much to the disadvantage of the latter. Moulden concludes that copper seems to have a more or less selective action upon Amœba coli as well as upon the lower forms of vegetable life, such as the algæ; that copper solutions are far better borne than those of quinine; that patients recover much more rapidly than under any other treatment known to him; and that the cure is permanent, provided the treatment is continued for a reasonable time after the disappearance of the organisms from the stools. As the patients treated number over 200 it is reasonable to conclude that the method is, at least, deserving of a thorough trial.

Thyminic Acid in Gout.—Dr. A. Breton (*La presse médicale*, 1906, No. 27, p. 211)) states that uric acid, according to the latest theories, does not circulate in the blood in its free state but in combination with thyminic acid, which is an amorphous brown powder, feebly acid, of insipid taste, and soluble in water. It is able to hold in solution an equal weight of uric acid at 68° F., and at body temperature this property is augmented by 50 per cent. The author concludes that gout is the result of a deficiency of thyminic acid in the serum, and he accordingly prescribes the former in this disease in doses of 10 grains per day. In eight patients such dosage has increased to a considerable extent, the output of uric acid and urea, the general condition has become ameliorated and the joints, previously stiff, have become much more supple.

Radium.—Robert Abbe (*Jour. Amer. Med. Assoc.*, 1906, xlviii, 183) concludes a paper upon the therapeutics of radium as follows: (1) The action of radium resembles that of the Röntgen rays. (2) It differs specifically and will cure some lesions which resist the latter. (3) It is applicable to the interior cavities of the nose and mouth, inaccessible to the Röntgen rays. (4) It is curative in most superficial epithelial cancers and lupus. (5) It has failed of curative action as yet in forty test instances of grave internal cancer. (6) It promises interesting results in other surgical conditions under study.

Heliotherapy in Psoriasis.—M. Guhr (*Berl. klin. Woch.*, 1906, No. 17, p. 536) has observed a cure of this obstinate cutaneous affection in a boy aged thirteen years, who was accustomed to take fresh water baths followed by lying in the sunshine, and has investigated the subject. According to him the treatment of psoriasis by means of sunlight is as follows: The patient's head being shaded, he exposes different parts of the body to the sun, the position being changed from time to time. The actinic rays act especially well in the spring when the skin has lost some of its pigmentation during the darker season. The duration of the exposure is from twenty to fifty minutes; gradually an abundant perspiration is set up, this may be increased by wrapping the patient, after a bath, in a linen sheet. Each exposure is followed by some hydrotherapeutic procedure, such as a Scotch douche or a partial bath, at the termination of which a promenade lasting a half, hour is taken. At first the treatment is followed by cutaneous sensations of heat, or itching; these disappear later. The author's experimentation seems to show that the treatment is of considerable practical value.

The Treatment of the Headache of Anemia.—Wilfrid Harris (*Practitioner,* 1906, lxxvii, 35) considers the headache of anemia to be due chiefly to the deficiency of hemoglobin, and consequent tendency to œdema, with the simultaneous starving of the meninges. It is usually frontal but may be vertical. In certain individuals of lymphatic type, subject to anemia, chilblains, and cold extremities, there may be a deficiency of calcium salts in the blood, and the administration of the calcium salts may be of great service in relieving the headaches of such patients. The lactate should be given in doses of 15 to 20 grains, three times a day. In this connection Harris states that the headaches of the morning after copious libations have been ascribed to a lack of calcium salts in the blood, these having been precipitated by the organic acids contained in the wine. This "toper's headache" may be quickly removed by a dose of 20 to 30 grains of calcium lactate shaken up with a little water.

The Use of Opsonic Factors in Therapeutics.—Ronald E. French (*Practitioner,* 1906, lxxvii, 64), in a paper upon the vaccine treatment of various infections, tuberculous, staphylococcic, etc., states that the ideal aim should be to obtain a cumulation of positive phases, and to avoid ever giving an injection during the negative phase. To do this it is necessary to take the opsonic index at regular intervals and to plot the results on a curve. The most suitable cases for injection treatment are those whose opsonic index is low and whose focus of infection is distinctly localized, for it is easier to raise a low opsonic index than to produce a rise in one that is already high. A tuberculous patient, who has an evening rise of temperature, should be kept in bed until the temperature is normal before inoculations should be begun. For inoculation, vaccines of known strength should be prepared, as the most satisfactory results are obtained from vaccines derived from the organism cultivated from the patient's own lesion, and standardized so that a given unit of volume shall contain either the dead bodies of a definite number of bacteria, or a definite weight of bacterial protoplasm. The vaccines in use at present are watery emulsions of bacterial cultivations diluted to contain some convenient number of bacteria, *e. g.,* 500,000,000 per c.c., sterilized by heating to 65° C. for an hour and finally sealed in capsules in doses of 1 c.c. For tuberculous patients, Koch's tuberculin (T. R.) diluted with 0.75 per cent. saline is used, previous sterilization being necessary. After the inoculation, which should be given with careful attention to asepsis, there is usually no local reaction. In a series of injections the first should be small; French gives as an initial dose 0.0002 milligram of T. R. or 250,000,000 cocci. If the reaction is satisfactory the dose need not be increased, but French recommends that it be doubled in adults in most instances. Too large dosage is indicated by a prolonged (two weeks or more) negative phase and by a lengthening of the successive negative phases. If the latter get successively shorter the dose is correct or too small; if it is too small the positive phases will not be well marked and the patient's progress will not be good. French, as a result of his observations, concludes that, although vaccine treatment stimulates the body resistance to infection, alone it will bring about recovery in but few instances. Other methods of treatment must be employed in connection with it. Surgical foci should be removed but not until the opsonic index has been increased, and it is strongly recommended that operations, particularly on tuberculous patients, should be performed only during the positive phase following a preliminary injection of tuberculin. In acute processes the inoculations are useless, but in chronic localized infections it is usually possible to increase the patient's resistance and aid nature to effect a cure.

PEDIATRICS.

UNDER THE CHARGE OF

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An Improvement of Kilmer's Abdominal Belt for Whooping-cough.—Such general satisfaction has been expressed by physicians who have used the elastic abdominal bandage originally suggested by KILMER, of New York, for the mitigation of the cough and vomiting of whooping-cough, that a simplified improvement upon the earlier design is well worth noticing (*Archives of Pediatrics*, February, 1907, p. 112). The original belt was composed of a long strip of silk-elastic webbing placed over a stockinette band; and this, while efficacious, was found to be too warm for comfort, besides being rather expensive. The new design is made of linen with a strip of elastic-silk webbing two inches wide inserted on each side. The belt laces in the back and is to be worn over the undershirt or band. The width of the band should be four to five inches for infants and five to eight inches for older children. The length of the belt should be three inches less than the circumference of the child's abdomen at the navel, thus allowing for a necessary amount of constriction when laced. If the first degree of constriction is not sufficient to produce a moderation of the cough and a complete cessation of vomiting the belt should be laced tighter. These belts can be made at a few hours' notice by any truss-maker, and should cost not more than from one dollar and a half to three dollars.

The Presence of Spirochetæ of Schaudinn in the Ocular Lesions of Syphilis.—In two heredosyphilitics, one stillborn and the other aged four weeks, SCHLIMPERT (*Deut. med. Woch.*, 1906, Nr. 36, p. 1452) has found spirochetæ in the conjunctiva, sclerotic and cornea, in the iris and choroid, and in the muscles of the eye and the lacrymal sac, confirming the opinion of certain authors upon the non-immunity of the lacrymal gland to the virus of syphilis. In both cases, but to a variable degree, the conjunctiva and cornea showed an infiltration of small cells, the choroid was congested and infiltrated, while the extrinsic muscles of the eye presented the lesions of myositis. Most frequently the organisms were found within the vessels and free in the blood. Schlimpert believes that this myositis, which he has found in his two cases, and which seemed to be determined by the spirochetæ, is the cause of the strabismus which is found so frequently with heredosyphilitics, reaching a proportion of 40 per cent. according to Fournier, or from 14 to 15 per cent. according to Huguenin.

The Assimilation of Iron in the Nursling.—KRASNOGORSKY (*Jahrb. f. Kinderheilk.*, 1906, xiv, 651) has studied the degree of assimilation of iron contained in human milk, in goats' milk, in spinach, in the yolk of egg, and in certain ferruginous preparations, such as ferratin. In all cases the iron was estimated in the substances studied and in feces and urine. The iron of human milk was absorbed in the proportion of 80 per cent., and 75 per cent. was assimilated. For goats' milk these figures were respectively 33 per cent. and 28 per cent. for the raw milk, and 15 and 9 per cent. for boiled milk. The iron of spinach is absorbed in a proportion of 64.2 per cent. and 61.5 per cent. is assimilated. The yolk of egg gives figures of 64.9 per cent. and 61.9 per cent. Finally for ferratin Krasnogorsky finds 23.78 per cent. for absorption and 23.39 per cent. for assimilation. The conclusion from this study, therefore, is that the iron contained in food is more completely absorbed and is retained in larger proportion than the iron of ferruginous preparations.

Changes in the Percentage of Neutrophiles during the Incubation of Measles.—According to the classification of ARNETH, the neutrophiles include six types according as their nucleus is single or composed of two, three, four, five, or even more lobes. In the normal state, in children, the percentage of these different forms averages 36 for the mononuclear type, 45 for the bilobed, 15 for the trilobed, and 3 for the multilobed forms. FLESCH and SCHLOSSBERGER (*Jahrb. f. Kinderheilk.*, xiv, 724), studying these forms during the incubation of measles, have found that these proportions are very characteristically modified from the figures obtained in health.

In 2 cases, four days before the appearance of Koplik's sign, they found the percentage of neutrophiles with simple nucleus increased to from 63 to 71, and attaining even 80 and 85 on the eve of the appearance of the buccal eruption. In 2 other cases, two days before the appearance of Koplik's spots, the proportion of these neutrophiles was 81 and 85 per cent. Finally, in a case in which the Koplik sign was absent, these cellular forms reached 60 per cent. on the fourth day before the exanthem appeared. This hematological sign appears, therefore, to possess a real value. It is, moreover, easy to observe, since it requires only an examination of the dried blood treated with the Jenner or triacid stain, without the necessity of a complete numeration. If found before the appearance of Koplik's sign, it would aid greatly in permitting the earlier isolation of impending cases, the value of which in prophylaxis has been already so greatly enhanced by the recognition of Koplik's discovery.

A Diagnostic Sign of Pertussis.—Referring to the difficulty in diagnosticating pertussis in very young infants in whom the cough is little characteristic and often difficult to provoke, TRIBOULET (*Revue mens. des maladies de l'enfance*, March, 1907, p. 138) describes a sign to which he attaches diagnostic importance. He forcibly depresses the base of the tongue, so as to see the lower part of the pharynx and if possible the tip of the epiglottis; the manœuvre often determines the enucleation from the pharynx of a globule of opaline mucus coming from the larynx, of a characteristic appearance not observed in any condition other than whooping-cough. He describes a case in which he was thus enabled to make a correct diagnosis based upon this sign. The infant was four months old, living in the country in an isolated farmhouse, from the immediate vicinity of which he had never been removed. No suspected person had come in contact with the infant, and the physician of the place asserted that no case had existed in the near or remote neighborhood for a long time. After the diagnosis was made it was discovered that the wet-nurse, who had been brought from Paris, had left her own baby, who had been suffering from the disease. In this case the direct mouth-to-mouth transmission of the disease had doubtless been effected through the intermediary of the wet-nurse's nipples.

Relapses in Measles.—E. Weill and L. Dauvergne (*Lyon méd.*, 1907, No. 3, p. 98) report an interesting example of two relapses of measles observed in an infant, aged five years. The primary attack occurred on May 15, 1906, and presented the classic picture of measles of moderate intensity. The eruption appeared normally, lasted four or five days, and then disappeared progressively. On June 13, the child, who had shown for six days a little evening elevation of temperature, presented a second eruption localized especially on the face, with a buccal exanthem, coryza, and lacrymation; the eruption lasted only thirty-six hours and was not so intense as the first. Between these two eruptions of measles the child presented an urticaria, six days after admission to the hospital, associated with fever which oscillated about 102° for twelve days. This eruption was attributed to a prophylactic injection of antidiphtheritic serum given on entrance to the hospital. About four days after the first relapse of measles the child again became feverish and the next day, June 19, a rubeolic eruption appeared, this time upon the face and thorax. In this second relapse the eruption was more intense, lasted longer (June 20 to 27), and was followed by a general furfuraceous desquamation. There was no angina, no abnormal rapidity of pulse, no albuminuria, nor any symptom suggestive of scarlatina, and the eruption was characteristically morbillous. Convalescence was normal and the child left the hospital perfectly well on July 18.

This observation appears to be conclusive as to the possibility of relapses in measles. The occurrence of two consecutive relapses is certainly rare, since Weill and Dauvergne have been able to find no similar case in the literature. In all the cases reported there was only a single relapse at a more considerable interval, ordinarily three or four weeks. The mechanism of these relapses remains here, as in other cases, purely hypothetical. It does not seem possible to admit an endogenous reinfection, such as is present in the typhoid relapse. It appears more reasonable to assume fresh inoculations, both because the organism, though unisolated, is known to have an ephemeral vitality, and because such relapses have been observed for the most part in hospitals or in large families where several children have the disease about the same time.

OBSTETRICS.

UNDER THE CHARGE OF

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Brow Presentation with Mentoposterior Mechanism at Birth.—LEO (*Ztschr. f. Geb. u. Gyn.*, 1906, lviii, Heft 1) reports the case of a primipara, aged nineteen years, who had a flattened pelvis. She came into spontaneous labor, when it was found that the anterior fontanelle was in front, and that the case was developing as a brow, the chin being behind. Under good uterine contraction the birth was spontaneous with the following mechanism: First, appeared the anterior corner of the right parietal bone, followed by the forehead and face, and then by the chin. Finally the occiput passed under the symphysis by the sinking of the head. The child was well developed, with normal measurements. The case is reported to show that posterior rotation of the chin is not always a positive indication that spontaneous labor cannot occur. Such cases as that reported usually happen when the cavity of the pelvis is capacious. Brow presentation results from flattening of the mother's pelvis, and also from relaxation and protrusion of the abdomen. The persistence of the brow presentation is observed because the child becomes fixed in the pelvis in this position. The fact that rotation does not occur is explained by the great distention and capacity of the soft parts. As normal children have been born with this mechanism the obstetrician should not immediately interfere in all cases.

The Cause of Labor.—KEHRER (*Archiv f. Gyn.*, 1906, lxxix, Heft 3) has studied the period of gestation in other than human animals. He finds that this varies from twenty-one days in the rat to six hundred and twenty-five days in the elephant. The duration of pregnancy is about the same in the human animal and the cow. In cold-blooded animals the duration of gestation, and the development of the young outside the womb, varies much more than among the warm-blooded. The duration of gestation corresponds to that period in which the young individual attains its greatest development. As probable causes of birth are mentioned the irritation of the uterus through mechanical pressure and the mature fœtus. Changes in the fœtal blood produce irritating substances which, passing into the blood of the mother, irritate the uterine nerves, producing contraction. At the latter portion of gestation there is a hyperemia of the genital tract which has rhythmic periods of increase. Labor usually occurs at one of these. Fœtal movements also seem potent in exciting labor.

The Treatment of Puerperal Septic Infection with Paltauf's Streptococcic Serum.—BURKARD (*Archiv f. Gyn.*, 1906, lxxix, Heft 3) reports 50 cases of infection treated by streptococcic serum. Paltauf's is a polyvant sérum, obtained from horses which have been given increasing doses of streptococci. In these cases the diagnosis was made by examining the lochia and the blood for streptococci. The serum was injected first into the infraclavicular spaces, and later into the anterior aspect of the thighs, in doses of 100 c.c. Patients also received intra-uterine douches of alcohol, while puerperal ulcers received applications with tincture of iodine and peroxide of hydrogen. The application of cold by packs and ice was also practised, but antipyretic drugs were not given. Vaginal douches were administered, as the effect of the treatment seemed to be to increase vaginal secretion. In about one-half the cases the patient suffered from an irritating and annoying urticaria. Some patients had pains in the joints. The eruption and pain usually subsided in five days or less after the injection. It could not be positively proved that the eruption was caused by the serum, as it was often seen in cases in which the serum was not employed. In 29 cases having pure streptococcic infection there were no deaths. In the whole series of 50 cases there were six deaths, two of them being mixed infection, two of streptococcic infection, one in which the bacteriological diagnosis was uncertain, and one in which bacteriological examination was not made. One of the cases was placenta prævia, frequently examined by a doctor and midwife outside the hospital, and found to have also purulent endometritis. After the injections of serum the temperature fell during the first twenty-four hours. In the second twenty-four hours it rose, probably from the absorption of the products of bacteria. A series of eight cases occurred in which bad results followed the injection, which could be traced to contaminated serum. It seems justifiable, from these cases, to conclude that the serum did good. To obtain the best results it must be used early in the case and promptly. When the case had been going on for some time, or when the infection was an overwhelming and fatal one, the serum had little effect.

The Function of the Kidneys in Pregnancy and in Eclampsia.—Buttner (*Archiv f. Gyn.*, 1906, xlix, Heft 2) contributes an interesting paper upon this subject. After prolonged examination of the urine, and analysis of the food taken by pregnant patients showing engorgement of the kidney and eclampsia, the author concludes as follows: The urine from the kidney of pregnancy, from normal patients, and during eclampsia, shows the characteristics of kidney insufficiency, and by cryoscopy resembles most the urine of kidney engorgement. The condition is not that of absolute suppression, but greatly impeded circulation of blood. In eclampsia this condition seems brought about by spasmodic contracture of the vessels, and probably the same condition pertains in the kidney of pregnancy and at the time of labor. Examination by cryoscopy shows that this condition is frequently present in normal pregnancy. The œdema which many patients show may be referred to this cause. In others the epithelium of the kidney becomes damaged. As a result of this the fluids of the body have a low specific gravity, which is typical of pregnancy, and seems to have a definite purpose. When œdema is present insufficiency of the kidney is usually absent. One of the important functions of the kidney seems to be to serve as a regulator of osmosis. As a result of this process the molecular concentration of the blood is greatly increased. The immediate cause of convulsions is found in arterial spasm in the brain.

GYNECOLOGY.

UNDER THE CHARGE OF

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Results of Atmocausis.—FUCHS (*Zentralbl. f. Gyn.*, 1906, Nr. 51, p. 1409) reports 163 cases, in none of which were any ill results noted; 88.2 per cent. of the cases were cured after resisting all other methods of treatment. In 44 in which the desired menopause followed the treatment no atresia occurred. The writer insists upon the importance of careful technique, especially the avoidance of metallic instruments. Curettement and thorough drying of the raw surface should always precede atmocausis. In order to secure obliteration of the uterine cavity exposure for a minute at a temperature of from 115 to 120° C. is necessary, the entire endometrium being subject to the hot steam from the fundus down to a point a quarter of an inch above the level of the os internum. In a few cases the slough may not be cast off for a long time, in which case it is only necessary to tampon the uterine cavity. Fuchs believes that the main indication for vaporization is climacteric hemorrhage.

Relation of Papilloma of the Ovaries to Papillary Cysts.—GOTTSCHALK (*Zeit. f. Geb. u. Gyn.*, Band lviii, Heft 2) dissents from the prevailing view that these neoplasms are entirely different in character. He cites the case of a nullipara, aged thirty-one years, from whom he removed a cystic ovary, the surface of which was covered with papillary bodies, while similar growths were observed on the inner wall of a cyst that had developed from the stroma. Microscopically the superficial papillomas were evidently outgrowths from the surface epithelium of the ovary, and were regarded by Gottschalk as the points of origin of the papillary cyst. All the papillomatous bodies, he thinks, might also have formed cysts were it not for the resistance offered by the thickened cortex.

Associated with the diseased ovary was a gonorrheal pyosalpinx, and it was interesting to note that the superficial papillomas developed at the point where the distal end of the tube was adherent, hence they may have been due to specific infection (?).

Hernia with Torsion of the Pedicle.—DAMIANOS (*Deut. Zeit. f. Chir.*, Band lxxx, Heft 304) reports a case of hernia in a child with symptoms of strangulation. On opening the sac it was found to contain a tube and ovary with twisted pedicle. He believes that the explanation of these cases (twenty of which have been recorded) is as follows:

The ovary in the newborn infant is easily pressed into the adjacent internal ring and is followed by the tube, no symptoms resulting unless a twist of the pedicle takes place, this being due, as Payr has pointed out, to a difference in tension between the relaxed tissues of the pedicle and its thin-walled veins, in consequence of which the latter dilate and wind around the resistant pedicle. The operation consists simply in removal of the adnexa. All the reported cases terminated successfully.

Curability of Uterine Cancer.—TAUFFER (*Gynaekologia; Zentralbl. f. Gyn.*, 1906, Nr. 51, p. 1412) contends that the early recognition of uterine cancer offers the only hope of permanent cure. From a review of 172 radical operations at the Budapest clinic between 1881 and 1904, he states that 44.05 per cent. of the patients kept under observation were free from recurrence at the end of five years. In from 8 to 10 per cent. death occurred in from three to five years after operation.

Malignant Neoplasms of the Tube.—ORTHMANN (*Zeit. f. Geb. u. Gyn.*, Band lviii, Heft 3) has collected reports of 84 cases of primary malignant disease of the tubes occurring in the course of twenty years, cancer being most frequent, next chorioepithelioma, and lastly sarcoma. He reports an interesting complication of primary cancer of the tube with malignant tuboövarian cyst. Of these 85 cases there was accompanying salpingitis in 44, which seems to support Sänger's theory of the inflammatory origin of tubal malignant disease. Secondary involvement of the tube in cancer of the uterus is exceedingly rare, the majority of the cases accompanying primary cancer of the ovary.

Observation of Fibroids by the General Practitioner.—WINTER (*Zentralbl. f. Gyn.*, 1907, Nr. 1, p. 26) calls attention to the fact that the indications for the surgical treatment of fibromyomas are furnished by the symptoms, degenerative processes, and complications. Severe pelvic pain not accompanying menstruation is indicative of some complication—in at least 10 per cent. of the cases of malignant degeneration. Vesical symptoms alone are not an indication for operation until other causes are excluded. It is especially incumbent upon the family physician to study the hearts of his patients with fibroids. Cardiac disease is rather an indication than a contra-indication for operation, though judgment must be exercised as to what risk must be taken. Winter believes that operations are indicated without symptoms in cases of subserous fibromyomas with long, slender pedicles, with rapidly growing tumors, and when they are of large size. The responsibility resting upon the general practitioner who has a case of uterine fibroid under observation is not light.

Re-implantation of the Ovaries.—PANKOW (*Zentralbl. f. Gyn.*, 1906, No. 44, p. 1224) reviews the results of experimental and practical work in this line in America and abroad, referring particularly to Morris' successful case of homoplastic transplantation. His own 7 cases of autoplastic and 2 of homoplastic transplantation lead him to infer that the former procedure is followed by definite results, which support the theory of internal secretion of the ovaries. The two latter experiments (in women who had been castrated two years before) were unsuccessful. Pankow makes the curious suggestion that the ovaries of newborn infants might be utilized in the homoplastic operation (!).

Vaporization of the Uterus.—Schaller (*Zentralbl. f. Gyn.*, 1906, Nr. 44, p. 1225) draws the following inferences from observations in a series of 26 cases: (1) Vaporization is a valuable aid to curettement in a certain number of cases. (2) It is impossible to regulate the dosage exactly because of individual peculiarities, so that unfavorable results must occur. (3) Hence vaporization should not be employed in the case of women of childbearing age, because obliteration of the uterine cavity or premature menopause may follow even a short exposure of the endometrium to superheated steam. (4) This method of treatment is absolutely contra-indicated in all cases of fibromyoma, since it is not only useless, but dangerous. (5) Exposure for six minutes to steam, even after thorough drying of the uterine cavity, often fails to produce the artificial menopause or obliteration; hence total extirpation is preferable in cases of intractable hemorrhage. (6) In some instances in which the most careful technique is adopted it is impossible to avoid partial obliteration, the leaving of portions of the mucosa, and the consequent formation of hematometra with resulting bad consequences. (7) Obliteration of the uterine cavity is nevertheless a valuable means of combating obstinate cases of uterine catarrh with accompanying pruritus when all methods of treatment have failed, providing that there is no infection present.

OPHTHALMOLOGY.

UNDER THE CHARGE OF

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The Importance of Bacteriology in Ocular Surgery.—Bach (*Ztschr. f. Augenheilk.*, 1906, vol. x) reviews the different sources of infection in the course of operations upon the eyes, the atmosphere, the hands of the operator, the conjunctival sac and free border of the lids, the instruments, the bandages. He attaches great importance to the conjunctival sac, which can never be sterilized perfectly; the same is true of the free border of the lid. He recommends scrubbing the surrounding skin with soap and mechanical disinfection of the free border of the lids with pledgets of cotton during irrigation with sterilized water. If the patient can be brought to the hospital the day before operation, it is well to apply a bandage, but this should be removed the next morning, but not immediately before the operation. Theoretically no bandage whatever is desirable after operation; but in practice many accessory factors make some sort of bandage necessary. Under the bandage, moreover, the tears continue to circulate within the conjunctival sac, contrary to what seems to be generally believed.

Experimental Researches upon the Visibility of the X-rays.—Bossalino's experiments (*Ann. di Ottal.*, No. 34, 1906) seem to show that the Röntgen rays are visible to the human eye in a condition of adaptation for obscurity; the presence of the crystalline lens is no obstacle to their visibility even when it is cataractous. On the other hand, it is indispensable that there be no pathological alterations of the fundus. The *x*-rays make visible the outlines of metallic objects interposed between the generating tube and the eye, and the visual perception is due to the retina, thanks to the fluorescence of the membrane itself.

School and Myopia.—Schnabel (*Wien. med. Presse*, No. 14, 1906) maintains that high degrees of myopia are neither caused by the school nor do they gradually develop from those cases of myopia which are acquired during school life. Such eyes are affected almost without exception with a grave anomaly of the ocular coat, posterior staphyloma of Scarpa, and this constitutes a fruitful source of danger to the structure and function of the retina. It is not the high myopia which is pernicious but the staphyloma, in which inheres, besides the myopia, the great probability of excessive danger to the function of the retina, and which accomplishes its work of destruction as thoroughly and as frequently in the case of manual laborers as of those who make large demands upon their vision. Moreover, myopias of medium degree (below 10 D. and above 6 D.) are never or but rarely caused by use of the eyes; in these cases also posterior staphyloma, retinal change, and diminished visual acuity are frequently found. Myopias of this degree include the highest degrees of myopia with healthy eyeballs and the lowest degrees of myopia from posterior staphyloma. But even myopia of lower and medium degrees (not above 6 D.) are not rarely found at an age before any demands have been made upon the eye, though in these cases the important influence of visual occupation upon the occurrence of the myopia is plainly evident; these are sound, normally developed eyes. Myopias of the highest grade do not develop from myopias caused by visual occupation; on the other hand, myopias so occasioned do not develop into myopia of the highest degrees. The school is not at all responsible for the misfortune which involves eyes affected with posterior staphyloma. It produces no diseased eyes, though it makes many myopic. It graduates many more myopes than it has received; but only the same number of cases of posterior staphyloma leave as have entered it.

Primary Tuberculosis of the Conjunctiva.—Gourfein (*Archiv. d'Ophtal.*, September, 1906) calls attention to the extreme rarity of tuberculosis of the conjunctiva, either primary or secondary. The statistics given by different authors, although differing greatly, prove it. Thus, Eyre counts one case in 3000 patients affected with ocular disease, Hirschberg one in 6000, Bock one in 10,000, Lagrange two in 15,000, Milligan one in 20,000, Mules one in 33,000. Gourfein himself has observed three cases in 49,000 patients treated at the Rothschild Foundation of Geneva during the past eleven years. The total number of published cases is 140 to 150 as a maximum, of which only eighty or ninety have been proved. As to primary tuberculosis of the conjunctiva only twenty-four cases are known in which the disease was proved experimentally or by the presence of the bacillus, and in which the integrity of the other organs was well established. These statistics are to be accepted with some reserve, as a few reported cases may have been overlooked. Gourfein gives a full report of two cases of the primary form, of which one, aged five years, died from cerebral tuberculosis, while the other, aged twelve years, was cured. His conclusions are (1) that tuberculosis of the conjunctiva can giverise to cerebral tuberculosis; (2) that the prognosis of the ulcerative form is graver than that of the other clinical varieties; (3) that primary tuberculosis of the conjunctiva may show lengthy remissions; (4) that primary tuberculosis of the conjunctiva is curable.

Squint, Its Causes, Results, and Treatment.—SCHOEN (*Centralbl. f. prak. Augenheilk.*, July, 1906) in a monograph of 250 pages considers the doctrine of squint from a standpoint decidedly different from the ordinary, as was to be expected from his former publications. The title "Hypertropia," etc., would characterize its contents still more exactly, the insistence upon the importance of vertical squint, to which the author has paid special attention for the past thirteen years, runs like a guiding line through the entire work. That vertical squint occurs has indeed never been denied in text-books and monographs, but it has been universally treated as a relatively rare occurrence. Increased attention appears to have been bestowed upon this condition recently in America, but no author probably has gone so far as Prof. Schoen, who finds in his private practice a vertical deviation, generally less, it is true, than 1 degree prism in 36.7 per cent. of all his cases. More astonishing even than this percentage is the extraordinary clinical significance attributed to this condition. Vertical deviation when of a degree impossible to be overcome without excessive asthenopia is declared to be the cause of an inexplicable convergent or generally divergent squint, in that the patient, to escape diplopia, prefers to sacrifice entirely binocular vision. In this way those numerous cases of squint for whose explanation all sorts of unproved hypotheses have been set up are to find their ready interpretation.

Vertical squint almost always dates from birth; it is to be ascribed to injuries received during severe and prolonged labor.

The nervous symptoms receive extensive consideration; these result from the excessive innervation required to prevent deviation of the eyes —to render the squint latent. In this connection also the more severe symptoms depend upon the innervation of the elevators and depressors. The accommodation and pupil are involved as well, but the muscular cramp radiates also to non-associated muscles. Vertical squint is declared to be an etiological factor in many cases of vertigo, insomnia, headache, acne rosacea, and chronic alopecia (?). In typical hemicrania Schoen finds ocular defects without exception, among which a vertical squint is very common, and he uniformly succeeds in curing the migraine by correcting the ocular error. The frequent syndrome called cardiac and gastric neurosis uniformly depends upon vertical squint, and the involvement of the vagus thereby occasioned; in fact, the whole conception of neurasthenia is almost to be identified with vertical squint. Finally, even chorea, hysteria, and epilepsy are brought into intimate connection with vertical squint; in diabetes (?) and cyclical albuminuria the connection is also suspected. If Schoen were correct in his conceptions, his researches would have to be looked upon as a scientific triumph of the greatest practical importance; for a large number of disease forms whose etiology is thus far but little understood, would at once receive their explanation and rational treatment. Meanwhile it will be well to preserve a neutral attitude and to notice whether and how far the very optimistic positions of the author will be substantiated by others.

HYGIENE AND PUBLIC HEALTH.

UNDER THE CHARGE OF

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Typhoid Fever and Shellfish.—An outbreak of typhoid fever due to polluted clams from flats near an outfall sewer is reported by J. F. BEALE (*Lancet*, 1907, i, 20): 7 persons ate of clams, which were said to have been thoroughly cleaned and boiled; 4 escaped with diarrhœa and vomiting, but 3 had typical typhoid. Clams obtained from the same source were found to be grossly polluted with sewage bacteria, which could be recovered alive from the tissues of the clams even after fifteen minutes' boiling. This result is in agreement with the findings of H. W. Clark ("Pollution of Shellfish," Thirty-seventh Annual Report of the State Board of Health of Massachusetts, Boston, 1906), whose tests with clams showed that one cannot be sure of sterility in all cases after five minutes' boiling, fifteen minutes' steaming, or by ordinary frying. The same was found to be true also o' oysters, which if cooked for more than five minutes are rendered unsuitable for eating, except that, by the method followed in the preparation of "escalloped" oysters, sterility may be secured after fifteen minutes' baking, without serious damage to texture and palatability.

According to a note in *Medical Record* (November 10, 1906), Wesleyan University, Middletown, Conn., has had a second outbreak of typhoid fever, traced to infected oysters consumed at frate nity banquets on October 12, 1906. (It was on October 12, 1894, that similar banquets were held, at which oysters fattened about 300 feet from a sewer outlet were eaten. More than twenty cases of typhoid fever occurred among the participants, but no others among the student body were seized, with one exception—a man who ate oysters from the same lot in the shop of the dealer.—C. H.)

The Paris correspondent of the *Lancet* (1907, i, 551) makes mention of 33 cases of typhoid fever in widely separated places traced to oysters from Cette. The cases were of a grave type and 7 resulted fatally. (Cette oysters are notoriously contaminated and unfit for food. Attention was drawn to their character in 1896 by Chantemesse, in 1900 by Mosny, and at intervals by other French writers.—C. H.)

Action of Boric Acid on the Bacteria of Meat and Sausage Poisoning.—In order to determine what action, if any, is exerted by boric acid on bacteria other than those which bring about putrefaction, which may be present in meat and meat products, R. BASSENGE (*Ztschr. f. exper. Path. und Ther.*, 1907, ii, 113) undertook a series of experiments with Bacillus botulinus and a number of related species, all of which have been isolated from meats which have caused outbreaks of poisoning, and also the colon and typhoid bacilli. The two latter proved to be unable to withstand contact with a 2.5 per cent. solution of the acid. In double this strength it prevented the development of but two of the meat-poisoning species; but even after six days' contact it was unable to destroy any of them. Thus the agent can constitute a real danger to health and life, since it can prevent decomposition by overcoming the slightly resistant bacteria of putrefaction, but cannot affect those which may cause poisoning. The meat products may appear to be of good quality, and at the same time they may be positively dangerous.

Poisoning by Canned Beans.—An unusually extensive outbreak of poisoning due to a most uncommon cause, namely, canned beans, is reported by ROLLY (*Münch. med. Woch.*, 1906, No. 37, 1798). About 250 employes of a single establishment were seized some hours after a free indulgence in canned beans with symptoms of acute and severe enteritis. In some instances the onset occurred about four hours after eating, and in about twenty hours many were brought to hospital for treatment. After from two to four days' disturbance the patients gradually recovered. The beans had been heated for a long time, but not to the boiling point, and most of the bacteria present must have been killed; but colon and paratyphoid bacilli were found. Broth cultures of the latter, type B., were fatal to mice and guinea-pigs after being heated to 100° C. for fifteen minutes. It appears, then, that the trouble was due to the toxins rather than to living bacteria. The paratyphoid organism was not found in the feces. (This is the second recorded outbreak of poisoning due to canned beans; the first occurred in Darmstadt in 1904, and was unusually severe, causing eleven deaths. The beans were not cooked, but were eaten as a salad. The symptoms were not those of enteritis, but of botulism, with the characteristic disturbance of vision and other evidence of involvement of the central nervous system.—C. H)

New Method for Formaldehyde Disinfection.—In place of the Flügge and other methods of formaldehyde disinfection, which are expensive on account of the amount of alcohol required, dangerous on account of the possibility of fire, and slow in disengaging the gas, EICHENGRUEN (*Ztschr. f. angewandte Chemie*, 1906, xix, 1412) advises the use of a compound of paraform and metallic peroxides (barium and strontium), which he calls "Autan."

It comes in the form of powder or in tablets, as desired. On being wet with water, a mixture of paraform and peroxides gives off formaldehyde more or less quickly, according to the proportions in which the ingredients are present. If they are present in equal amounts, the powder on being mixed with twice its weight of water explodes with violence; but as prepared it gives off the gas with comparative slowness, but yet sufficiently rapidly. SELTER (*Münch. med. Woch.*, 1906, liv, 2425) states that the action is so rapid, and the room is filled so quickly, that it is hardly ncessary to do more than close the larger ventilating openings. In his experiments, using as test objects staphylococci, anthrax spores, and fresh tuberculous sputum, the results were good, although a few staphylococci and many anthrax spores survived. Wesenburg (*Hygienische Rundschau,* 1906, xvi, 1241) conducted a series of experiments, in which he used as tests old library books, Staphylococcus aureus, anthrax spores, colon and typhoid bacilli, and pus (Staphylococcus aureus and Bacillus pyocyaneus), with good results. He used an ordinary kettle, in which he placed 1000 grams of autan, upon which he then poured 900 c.c. of water. Not only was the gas given off rapidly, but the air of the room was quickly saturated with aqueous vapor. From his results he concludes that complete disinfection may be expected from the use of 1.4 kilos of autan per 50 cubic meters of air space, with exposure from six to seven hours. He recommends the process as particularly adapted to the disinfection of closets, wardrobes, and other small spaces, with their contents. It is perfectly safe and very simple, there being no danger of fire and no need of any apparatus, beyond a pail or tub and a stick with which to stir the mixture with water. The material is put up in tin cans for rooms of different cubic capacities, and each can is accompanied with an "ammonia generator" containing ammonium chloride. The strongly alkaline residue in the can is treated with water, the ammonium salt is poured into it, and the resulting ammonia neutralizes the formaldehyde gas after the desired period of exposure.

With the suggestion of Eichengrün, Selter, and Wesenberg, that it is unnecessary to make a room as airtight as possible, A. Nieter (*Hygienische Rundschau,* 1907, xvii, 151) does not agree (and how can anybody?). Nieter finds the advantages of the process to be its simplicity, the freedom from danger of fire, and the possibility of a number of disinfections going on simultaneously under control of a single official. On the other hand, a distinct disadvantage is the high price of the material. (From the literature on the subject, it is difficult to learn wherein this process possesses any advantage over the equally simple and safe permanganate and formalin method of the State Board of Health of Maine.—C. H.)

A New Disinfectant: Metakaline.—Metakaline, a compound of hard soap and potassium metacresolate, is found by G. Wesenberg (*Centralbl. f. Bakter.,* 1907, xxxviii, 612) to possess great germicidal power. In 0.5 per cent. solution it is effective against the common pathogenic bacteria within a few minutes, and is absolutely non-irritating.

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THE

AMERICAN JOURNAL

OF THE MEDICAL SCIENCES.

JUNE, 1907.

ORIGINAL ARTICLES.

TUBERCULIN IMMUNIZATION IN THE TREATMENT OF PULMONARY TUBERCULOSIS.

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The specific treatment of tuberculosis by tuberculin is based on the principle of artificial immunization. The production of artificial immunity to tuberculosis has been long looked upon as impossible of attainment, because there is little clinical evidence that one attack protects from another. Nevertheless, the tireless and painstaking experimental work which has been carried on by many observers during the past fifteen years has done much to demonstrate the possibility of producing, artificially, in animals a certain degree of immunity. This immunity has been shown to be only relative, and the best results have been invariably attained thus far by vaccination with living bacilli, while the inoculations of culture products freed from germs or of bacteria killed by heat or other methods, though they have been found to be of some value, have never brought about as good results as vaccination with living germs. It is to the study of the mechanism of artificial immunization in animals that we must look to help us formulate a method of attempted immunization in the tuberculous patient; but unfortunately the mechanism of artificial immunization against tuberculosis, like that of all chronic infectious diseases, is as yet but very imperfectly understood, though our knowledge of the many defensive resources of the living organism against the toxin and the microbe itself is steadily growing. We know that there is an antitoxic immunity, an antibacterial immunity, and an immunity produced by vaccination with living bacteria which follows as a result of the victory of the tissues over the living germ, and to which von Behring has given the name of isopathic immunity. The exact part played by the antitoxins, by the various antibodies, the agglutinins, the bacteriolysins, and the opsonins, is as yet imperfectly understood; but we may take if for granted that these are all part of the mechanism of immunization and factors in Nature's attempt to protect the body against bacterial infection. Wright's method of immunization accepts as the sole guide for dosage and intervals between doses one of these immunity reactions, as demonstrated by the opsonic index. One thing we may learn from the more recent studies on the production of artificial immunity to tuberculosis is that the dose of virus, whether living or dead, and the time which elapses between the protective inoculations and before exposure to reinfection, are essential elements on which success or failure may turn.

Calmette, in his recent studies of vaccination of calves and young goats through the intestinal tract by the introduction of living virulent tubercle bacilli, found that whether a localized process limited to the mesenteric glands and ultimately resulting in a marked degree of immunity or general infection resulted, depended entirely on the number of the bacilli introduced and the time which elapsed before another infection. A very small amount of infectious material becoming arrested in the intestinal and mesenteric glands, and attacked there by the leukocytes resulted in immunity to subsequent inoculations—inoculations which prove fatal to control animals—while a larger amount, or repeated inoculations of the same small dose, though arrested for a time in the mesenteric glands, passed on through the lymph stream into the circulation, and ended in general infection and the death of the animal.

Von Behring has found practically the same thing, and the work of Wright and Douglas on the opsonins shows that even when dead culture products are made use of, the size of the dose governs the result, which may be a prolonged negative phase with its decrease of the natural resisting power, or a positive phase resulting in a relative degree of immunization. If we use tuberculin by the clinical method we notice the same thing; namely, that infinitesimal doses, methodically increased, which produce only slight stimulation of the defensive resources of the organism, result in a well-marked degree of toxin immunity, as shown by increased toleration to large doses of toxin, while larger doses or too rapid an increase, may bring about hypersusceptibility and an aggravation of all the symptoms of the disease.

The importance of time in the production of artificial immunization has also been thoroughly demonstrated. It seems that whatever degree of immunity it is possible to produce is produced only very slowly. Von Behring found that his vaccinated cows which received the virulent inoculation before three months had passed showed little immunity and generally died of the infection, while after three months they resisted a fatal dose of the virus. Experimental evidence, therefore, teaches that the dose of the vaccine and the time occupied by the treatment, as well as the time which elapses between doses, must prove cardinal points in any attempted method of inoculation with bacterial products, and clinical study of tuberculin treatment in man leads to the same conclusion. While the principle of artificial immunity seems to be fairly well established by animal experimentation, it must be admitted that the laboratory evidence which bears on the production of immunity in animals, or the cure of experimental tuberculosis by tuberculin, is far from satisfactory. Even Koch has failed to give the details of the experiments in which he claims to have protected guinea-pigs against infection by treatment with his two immunizing tuberculins, T. R. and B. E., and he contents himself with simply stating that this can be done. I know of no one else, however, who has cured experimental tuberculosis in the guinea-pig, and I have always failed to do so myself. Behring, like Koch, claims to protect guinea-pigs, but gives no facts.

The best results I have obtained in treating animals with tuberculin have been shown in the eye tuberculosis of the rabbit, which is naturally a chronic and almost always a purely localized process. Tuberculosis in the guinea-pig, on the other hand, is an acute progressive infection, and experimental and clinical evidence are in perfect accord in demonstrating that against the acute types of tuberculous infection tuberculin is powerless, whether it be employed in man or animals.

The duration of artificial immunity, such as has been successfully produced so far in animals, has not yet been ascertained, but the evidence so far at hand points to the fact that as the most solid immunity is produced by living though attenuated cultures, the immunity which lasts the longest is also brought about in this way, the antitoxic immunity produced by bacterial products being of much shorter duration. This is perhaps not as discouraging as it might at first sight appear, because in our therapeutic attempts at immunization with dead bacterial products the patient already has within his tissues the living organism, the victory over which seems so essential to the production of the most solid immunity, and if by properly spaced and properly graded inoculations of bacterial products we may assist him to overcome his infection, the immunity thus obtained might be quite durable.

Tuberculins. Koch, to whom we owe the discovery of tuberculin, was also the first to propose its therapeutic employment. Curiously enough, he did not at first consider his old tuberculin as an immunizing agent, but thought the local reactions it produced resulted in a necrotic process which ended in the discharge of the dead and diseased tissue and healing of the lesion. His original tuberculin is made by evaporating by heat liquid cultures to a tenth of their original volume, and filtering off the bacilli through a porcelain filter. This tuberculin, which he called a glycerin extract of the tubercle bacillus, is the one which is used for the tuberculin test both in man and animals, as well as therapeutically. His two more recent tuberculins, T. R: and B. E., represent attempts at producing antitoxic and antibacterial immunity, and are suspensions of crushed tubercle bacilli in glycerin and water. Many other tuberculins have been proposed during the past fifteen years, among which I may mention Hunter's Modification B., von Ruck's Watery Extract, Landemann's Tuberculol, Denys' B. F., Baraneck's Tuberculins, Spengler's Bovine B. F., and Behring's T. C. and Tulasa, which he claims immunizes cows as well as the living bacilli, but the value of which has not yet been put to a practical test in the treatment of human tuberculosis. These tuberculins are all vaccines, they are all made from either the body substance of the germ or the liquid medium in which it has grown, or both, and their aim is to stimulate the defensive resources of the system and to induce antitoxic and antibacterial immunity. They all produce, when given in sufficient doses, local reactions in tuberculous foci, and the well-known but little understood phenomena of general tuberculin reaction.

Koch's old tuberculin principally has been used at the Adirondack Cottage Sanitarium, but of late I have discarded it, because I prefer an immunizing agent in the preparation of which no heat has been used, and I make use of either B. F., which is merely a filtrate of human cultures of recorded virulence grown in my own laboratory, to which a quarter of 1 per cent. carbolic acid has been added, or B. E., which is an emulsion in glycerin and water of the pulverized bodies of virulent tubercle bacilli. The first, which is known as Denys' tuberculin, and for which he has a strong preference, contains all the toxins and other substances which may be produced by the growth of the bacilli or dissolved from their disintegrating bodies in a liquid medium; and the second, which is Koch's last attempt at producing an immunizing substance, contains the toxins and endotoxins to be found in the bodies of the crushed germs entire and unaltered by heat or chemicals.

The use of B. F., from bovine cultures, was proposed by Spengler, and it is possible that in infections of bovine origin it may be preferable to tuberculin made from tubercle bacilli of human origin. I am applying it in a few cases in which the patient does not seem to be making any improvement while treated with B. F. of human origin.

Reasoning from analogy and what we know of artificial immunity in other bacterial infections, an antitoxic immunity would be more likely to be produced by the old tuberculin and the B. F., which contain the toxins elaborated by the growth of the germs in a liquid medium, while the B. E., which holds in suspension the crushed body substance of the tubercle bacilli, might be expected to produce a greater degree of antibacterial immunity.

I am not a partisan of any particular tuberculin, but I use the B. F. at present more extensively than the B. E., only because I am more familiar with its use, and because it is easier to control in its effects. With the B. E. habituation takes place with much more difficulty, and occasionally unexpected and sometimes violent reactions occur, even if the utmost caution in increasing the dose is exercised. It is possible that having obtained a certain degree of antitoxic immunity with a course of B. F., a secondary course in which B. E. is employed might prove more efficacious, and it is evident we have much yet to learn about the production of the tuberculous vaccines and their application in the treatment of disease. Time and experience alone can show us which tuberculin produces the best results, but thus far it has become apparent to me that whatever good results may be obtained depend quite as much upon the method used by the physician who administers the tuberculin as upon the kind of tuberculin he administers.

The method is of the utmost importance. We have seen that success or failure in attempts at immunization experimentally depends to a great extent on the method of immunization, and this must hold with even more force in trying to produce a certain degree of immunity in the individual already suffering from an infection. We have at present two methods to guide us in the use of tuberculin: the laboratory method and the clinical method. The laboratory method is that which has been put forward by Sir Almaroth Wright as the result of his discovery of opsonins, and relies solely on the readings of the opsonic index as a guide, both as to dose and to intervals between doses. What may be called the clinical method has been developed gradually from the experience of those using tuberculin, and by it the dose given, the interval between doses, the length of the treatment, and the ultimate dosage reached, are controlled entirely according to the clinical manifestations of the case and the effect of the injections on the patient's condition and symptoms.

Little experience has as yet been accumulated in the treatment of tuberculosis, especially pulmonary tuberculosis, by Dr. Wright's method of using bacterial vaccines, but excellent results are reported in localized tuberculosis. It is greatly to be desired that we may learn something from Dr. Wright's discovery that will place the treatment of tuberculosis by vaccines on a more definite and scientific basis, and show us with more exactitude when our injections are benefiting the patient and also when they may be doing him harm. Apart from the technical difficulties involved in taking the opsonic index of a large number of patients under treatment, the great amount of labor, skill, and time necessary in following out such a procedure in each case, and the numerous sources of error which militate against accuracy, we will have to learn more definitely how reliable a criterion of immunity the degree of phagocytosis really is.

The production of opsonins, like that of agglutinins, precipitins, etc., is only part of the complicated mechanism of resistance put forth by the living organism, and its exact meaning as a criterion of immunity needs to be more definitely demonstrated. Although in a general way increased phagocytosis means increased immunity, we must remember that by itself this reaction does not necessarily imply that the system putting it forth is immune. The englobing of the bacilli does not necessarily mean their destruction. We find in guinea-pigs, for instance, that if tubercle bacilli are introduced into the peritoneal cavity they are taken up in large numbers by the polynuclear leukocytes and englobed, and yet these animals are most susceptible to tuberculosis, and the englobing of the bacilli does not in any way protect them against general infection.

The old clinical method, which was tried with such disastrous results in the earlier history of tuberculin treatment, was based on the production of pronounced local and general reactions which were thought essential to cure. These we know now to be harmful, and the avoidance of marked reactions and the production of tuberculin immunity are the main features of the new method. The clinical and laboratory methods are in perfect accord as to the efficiency of minute doses and the danger of over-dosage; but Dr. Wright's method takes no account of habituation by progression in dosage and the production of tuberculin immunity, and merely aims at keeping the patient's opsonic index as high as possible by suitable and properly spaced small injections of the same, or about the same, amounts of tuberculin indefinitely repeated.

The clinical method aims to carry the patient to large doses—doses one hundred, one thousand, ten thousand times larger than the commencing dose—namely, to produce tuberculin immunity while avoiding marked reactions or any disturbance of the patient's general health; this may be accomplished in most cases by beginning with minute amounts, increasing so gradually and at such intervals, and extending the treatment over as long a time as is necessary to accomplish the desired result, no matter how long that may be. The main difference in the two methods, therefore, is that of progression in dosage, and also, to a certain extent, the interval between the doses, which is much longer in Dr. Wright's method than by the usual clinical method; and the question resolves itself into this: Which is the most reliable guide to dosage—the opsonic index, or the clinical observation of the patient's condition and symptoms? And is the production of tuberculin immunity essential or not?

In favor of progression it may be said that whatever effect we may produce by simply repeating indefinitely the same small dose which maintains the opsonic index above normal, by progression in dosage we certainly bring about in the infected organism a very self-evident and easily demonstrable change; a change which renders it unsusceptible to doses of tuberculin sometimes ten thousand times greater than the amount which at first would have produced marked evidence of intolerance and violent constitutional disturbance, an amount many times larger than the healthy individual can withstand. In bringing about tuberculin immunity have we depressed the opsonic index, produced long negative phases, and harmed the patient? or have we progressed so gradually as to avoid negative phases, and has the stimulation of the cells resulted in the formation of antitoxins or antibodies which protect the infected individual, at any rate against some of the evil influences of his infection?

The improvement in the patient's general condition and in all the symptoms which are the direct result of his chronic toxemia while being injected with increasing amounts of toxin, would point to the conclusion that whatever influence progression in dosage has on the opsonic index, it has a favorable influence on the patient's disease so long as it is administered within the limits of his power to respond to the increasing doses by the production of increasing amounts of antitoxic or protective substances.

Are we in future to disregard progression and antitoxic immunity and confine ourselves purely to raising the opsonic index by using repeatedly the same small doses at long intervals?

Even if we are not, it is to be hoped that Dr. Wright's work may teach us where the clinical method is at fault, and how to remedy its defects. Any physician who has used tuberculin therapeutically very soon learns that it is an agent powerful both for good and for evil; that he is working somewhat in the dark; that he may at times be giving too much and at times too little; and that he needs some more accurate method to guide him in regulating the doses and the intervals between injections. Even in the present state of our knowledge about tuberculin immunization, however, this much I think may be said: that though better results might be had if we possessed more accurate data as to what we are accomplishing through our treatment, nevertheless if the rules which experience has already taught us are carefully followed there is little or no danger from tuberculin treatment by the modern clinical method. It is the gross disregard of these rules which may bring about disaster and aggravate the patient's condition. The main object to be kept in view in tuberculin treatment by the clinical method is to produce as strong an immunity to tuberculin as possible, while frequently repeated, mild and hardly perceptible local reactions in the tuberculous foci are taking place, but without bringing about any general fever reactions, violent local reactions, or disturbing the patient's general health more than is absolutely unavoidable. Physicians who have continued the tuberculin treatment have all tried to meet the indications I have just referred to in their own way and in the light of their own personal experience, and from their observations the present mild method has been gradually evolved.

In the therapeutic use of tuberculin it is best to have a certain formulated schedule by which the initial dose, the intervals between injections, the rate of progression, and the ultimate dose to be attained are distinctly laid down, this schedule to be literally followed so long as the patient shows no evidence of intolerance, but modified at once, as soon as he does, to suit the requirements of each case. Many patients can be carried from beginning to end of the treatment—a period which, when no reactions occur, usually takes about eight months—without any symptoms which call for any departure from the schedule laid down. If this were always so the treatment would be simplicity itself, but unfortunately in the majority of cases, at some period in the treatment, sometimes at the very beginning, sometimes at the middle, and sometimes even at the very last dose, symptoms of intolerance appear, and it is then that the physician requires certain definite rules to guide him in his conduct of the case, and these rules I will briefly touch upon.

Each physician modifies any schedule of dosage he may use according to his experience as he becomes more familiar with the effects of the powerful toxin he is employing. My own ideas as to certain rules have grown into convictions, or have little by little undergone changes as my experience grew. At first I formulated a schedule of dosage of my own, which, however, lacked definiteness; but of late I have adopted the plan of injections laid down by Denys, because it has seemed to me simple, definite, and, so far as I have used it, practical, and can be easily altered to meet the requirements of each case, though it is no doubt susceptible of improvement.

The first rule—one which practical experience has long ago taught me to be essential—is to begin with very small doses; much smaller doses than are usually advised. It we remember that the object of tuberculin treatment is to avoid constitutional disturbance and violent reactions which lead, especially if repeated, to tuberculin hypersusceptibility and aggravation of the patient's symptoms, we will soon realize that the best way to do this is to build up the tolerance to the toxin very gradually; in my experience, by following this rule, the number of cases which go through the whole treatment without marked reactions is increased. Our aim should also be to put off reactions as long as possible, because if a marked reaction occurs at the beginning of the treatment it is exceedingly difficult to habituate the patient to increase in the dosage, while later in the treatment, possibly owing to the partial immunization already produced, reactions are more rarely followed by evidence of prolonged intolerance. It has been amply demonstrated that tuberculin hypersusceptibility may be produced in cases in which the diagnosis had been made only by the tuberculin test, so that the one marked reaction which the test had developed made the individual, slight as the disease was, for a long time so hypersusceptible to the toxin that it became exceedingly difficult to produce any tolerance when the treatment was begun.

We find also that reactions take place more readily at doses fixed between 0.1 milligram and 1 milligram than perhaps at any other stage. We should, therefore, begin sufficiently far down the scale to carry the patient through these doses without producing a reaction. For all these reasons it is essential to begin treatment with very small doses; that is, for afebrile cases, $\frac{1}{10000}$ milligram of filtrate B. F., or Koch's B. E. (liquid measure, not solid substance), or $\frac{1}{1000}$ milligram of old tuberculin. Denys makes use of eight solutions in giving B. F. No. 1 contains $\frac{1}{10000}$ milligram to each cubic centimeter. This is for febrile cases only. No. 2 contains $\frac{1}{1000}$ milligram to each cubic centimeter; No. 3, $\frac{1}{100}$; No. 4, $\frac{1}{10}$; No. 5, 1 milligram; No. 6, 10 milligrams; No. 7, 100 to each c.c., and No. 8 is pure filtrate. Now the increase in using these solutions is always by 1 decigram of each solution, which is convenient to measure and easy to remember. As 10 decigrams, or 1 c.c. of each solution is reached, the next solution, which is ten times stronger and in which 1 decigram represents the same dose as 1 c.c. of the preceding solution, is taken up and the increase is again by 0.1 of the new solution until 1 c.c. is given, when the next solution is taken up in the same way until the end of the treatment. Thus for ten doses the increase for each dose is by $\frac{1}{10000}$ milligram for ten doses; then by $\frac{1}{1000}$ for ten doses; then by $\frac{1}{100}$ for ten doses; then by $\frac{1}{10}$ for ten doses; then by 1 milligram for ten doses; then by 10 milligrams for ten doses; then by 100 milligrams, until 1 c.c. of the pure filtrate or old tuberculin is reached. The increase is by 0.1 of each solution, and as each solution is ten times stronger than the preceding, the progression in doses is ten times greater at the end of every ten doses. Certain details of the treatment may be gathered from the appended Charts I and II. Approximately the same plan may be followed in giving Bacillen-emulsion, provided it is remembered the doses referred to in the above schedule are liquid measure and not solid substance.

Dr. Brown has found that at the Adirondack Cottage Sanitarium reactions occur more frequently at the second or third injection of a new solution. This is not to be wondered at, as the increase in dose is ten times larger when a new solution is taken up. To obviate this, the progression for each solution can be by 0.5 decigram instead of 1 decigram for the first three doses; then by 1 decigram for the remaining doses in each solution, or, if time is to be considered, the amount taken off the first three or four doses can be added in suitable proportion to the last six or seven. Thus instead of increasing 0.1, 0.2, 0.3 and so on to 10 decigrams the increase may be 0.1, 0.15, 0.2, 0.25, 0.3, 0.4, 0.6, 0.8, 0.10. In truly apyretic cases with good nutrition the physician in his discretion can make the increase for the first two solutions by 0.2 decigram, but after the dose of $\frac{1}{100}$ milligram is reached the increase for the remaining solutions should be 0.1 decigram of each solution.

The intervals between the injections are three or four days; generally two injections a week; but as the higher doses, such as 10 milligrams, are reached, the intervals may be five days, and after 100 milligrams six days, while the last three or four doses should be given a week or ten days apart. Lowenstein finds that for Bacillenemulsion longer intervals are necessary between the doses, especially when the large amounts are reached.

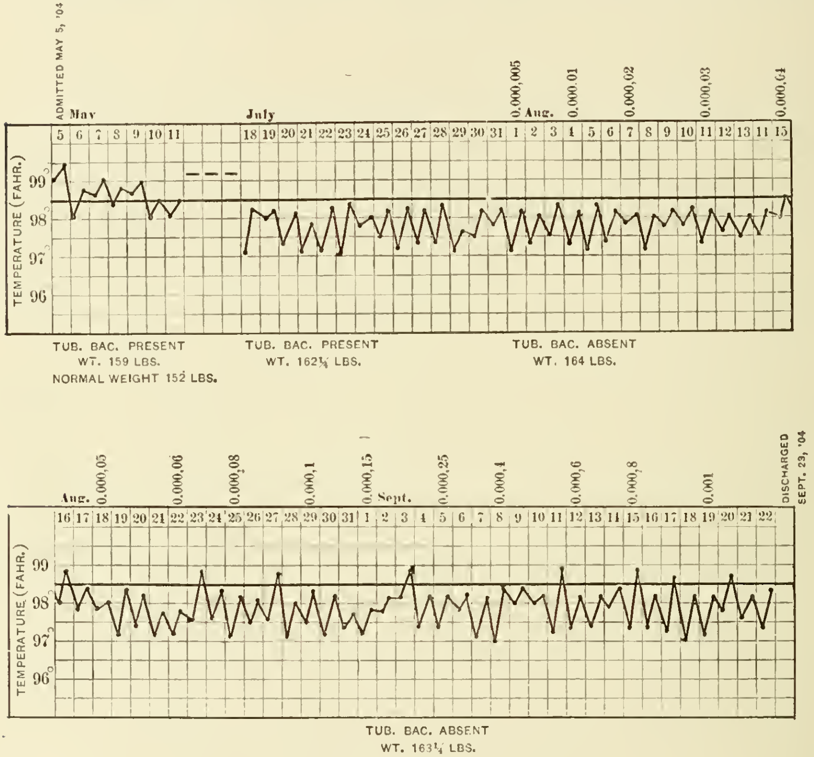

Fig. 1.—Details of tuberculin immunization.

If no intolerance is manifested the treatment will require six months; but in the majority of cases when any reactions occur it should be extended over ten months or a year, or even much longer, if necessary, to reach full doses. It is a mistake to try to shorten the time by increasing the doses too rapidly, or decreasing the intervals. Whatever degree of immunity, antitoxic or otherwise, is produced by the treatment is produced only very gradually, and besides the risk to the patient which is always involved by haste, the intolerance it may produce takes often so long to overcome that the duration of the treatment is lengthened rather than shortened in the end.

Tolerance to tuberculin is an excellent prognostic sign and it bears a certain relation to the condition of the patient's general

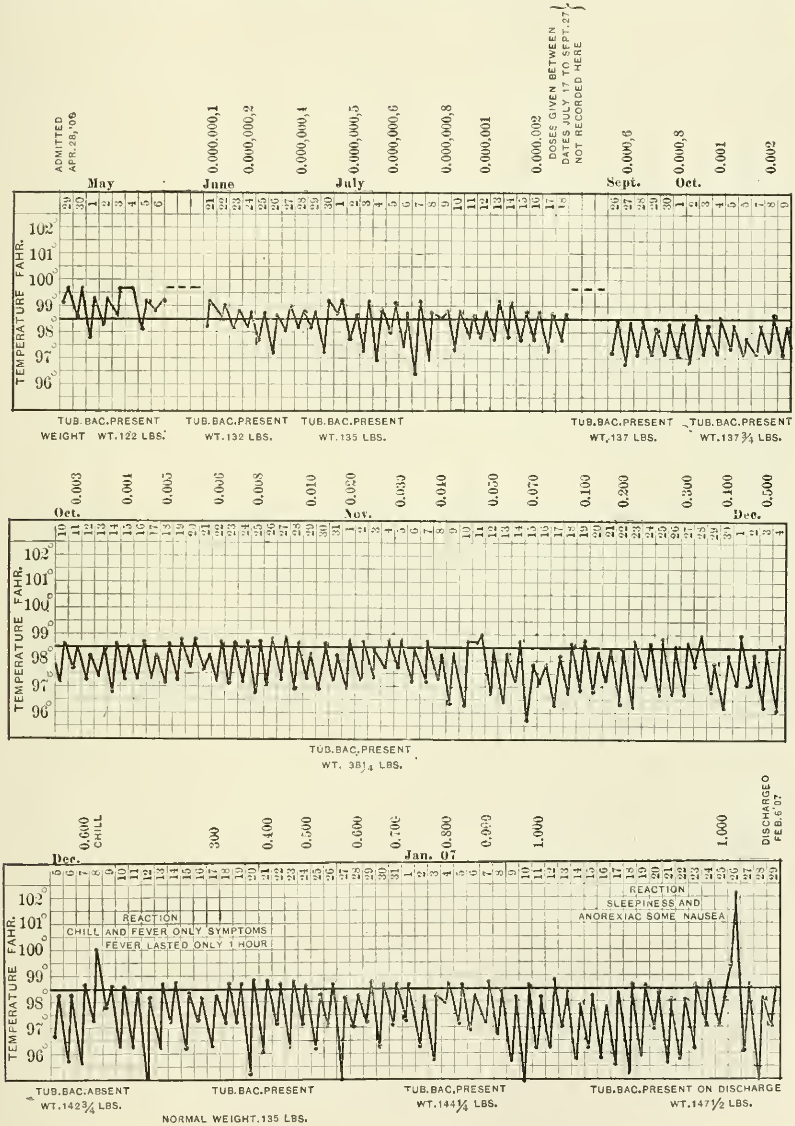

FIG. 2.—Details of tuberculin immunization.

health; and the more this improves the less apt is he to develop symptoms of prolonged intolerance, but the improvement in the general health is necessarily a slow process.

How does intolerance show itself, and how are we to proceed when it does? The symptoms of intolerance may be divided into three groups: those of a general fever reaction, those which indicate local reaction, both at the site of disease and also the site of injection, and those which point to general constitutional disturbance, as manifested by malaise, headache, sleeplessness, wandering pains, anorexia, nausea, and loss of weight and strength. Any one of these three groups of symptoms which constitute the fever reaction, the local reaction, or the constitutional impairment may be present separately, or some of the symptoms out of each group may show themselves; but they all, whether present separately or collectively, indicate intolerance and are to be considered before another injection is given, and in determining its strength when it is given.

The fever reactions are of two kinds: the short and the prolonged reaction. The short reaction is identical with that produced by the tuberculin test, and shows the classical fall and rise of temperature all ending in forty-eight hours; the prolonged reaction begins generally more gradually; the symptoms are mild; the fever rises less high but maintains itself, with a morning remission, above the patient's normal temperature range for several days, generally not more than a week. These prolonged reactions show the same temperature curve and all the other manifestations so commonly observed in the exacerbations of chronic pulmonary tuberculosis, except that usually they are of shorter duration. The temperature may not be more than 1° above the patient's usual range, or even less, but it does not fall for several days.

Local reaction at the site of the lesion is a valuable guide to dosage; increased cough and expectoration, pleuritic pains, aggravation of the physical signs, hoarseness, pain and aphonia if the larynx is involved, all point to local reaction and are all indications for caution in increasing the dosage. If moderate, they soon subside if the injections are discontinued for a short time or the dose lowered, and, no doubt, when moderate, local reactions are of benefit to the patient in bringing about reparative processes and in ridding the system of pus and septic material, for we have no better expectorant than tuberculin. In support of this view is the improvement in the patient's general condition and the marked diminution in the fever, cough, and expectoration which often follow moderate local reactions at the site of the lesions.

Local reaction at the site of injection shows itself by more or less extensive redness, œdema, and pain at the site of the injection; when slight, it may be disregarded, as it is somewhat influenced by the manner of injection or other causes; but if marked, it indicates commencing intolerance and should be considered in connection with the patient's other symptoms before increasing the dose.

Most important, and most often disregarded because no rise in temperature may be present, is the group of symptoms which point to constitutional impairment resulting from overdosage. They are all the symptoms that chronic toxemia might be expected to produce, and all point to the supposition that the patient cannot respond by the formation of antitoxins and antibodies to the increasing doses of toxin which he is receiving. Even if no fever above the usual range be present, the patient who has been improving and whose general condition has been satisfactory, may show marked arrest in his improvement; if the injections are persisted in and the dose steadily increased, he will complain of malaise, exhaustion, headache, sleeplessness, wandering pains, anorexia, nausea, and loss of weight. If these symptoms are disregarded, the injections continued, and the dose heedlessly increased, in time the patient's disease may take on an acute form. When intolerance manifests itself, whether by general fever reactions, by evidence of local reactions, or by some of the symptoms of constitutional impairment, the rule is never to inject while any of these symptoms is still present, but to wait until the temperature has returned to its usual height, until the cough and increased expectoration have lessened, and all evidence of constitutional impairment, such as anorexia, malaise, debility, etc., have disappeared. Indeed, all evidences of intolerance must have been absent for at least two days before the injections are again taken up. When a febrile reaction has occurred, if the fever has been high or prolonged and the constitutional disturbance marked, the same dose that produced the disturbance should not be repeated, but should be decreased by one-half or more; if this produces no ill-effect the usual ratio of increase and intervals may be resumed until the dose which produced the reaction is accepted without disturbing the patient. If the reaction has been very mild the same dose which produced the symptoms may be repeated, but it is usually better to decrease the amount somewhat. In case local reactions at the site of injection or at the site of the lesions takes place without other symptoms, an interval of rest may be given and the same dose repeated. Sometimes it happens that there is no distinct fever reaction, no distinct local reaction, but as the doses are increased the patient begins to show some of the symptoms of constitutional impairment referred to; the same principle would apply here: discontinue injections until the unfavorable symptom or symptoms have disappeared, then begin again with greatly reduced doses, and increase gradually as heretofore. When in doubt as to the explanation of any slight symptom of intolerance, always omit one or two injections and then begin by repeating the last injection.

We have learned that "No reaction, no cure," has been a most misleading axiom, for we can have tuberculin immunity without reactions, and many reactions without any tuberculin immunity. Strong and frequent reactions are harmful, and yet while patients who go through the entire treatment without appreciable fever reactions derive all the benefit that could be expected from the treatment, I have noticed that often when the patient's improvement seems at a standstill he will again begin to improve after he has had some evidence of a moderate local or even general reaction. The disturbance caused from the reaction having passed away, injections having been discontinued for a while and the dose reduced, the improvement in the general condition and symptoms seems to manifest itself anew when before it was at a standstill.

Tuberculin hypersusceptibility is an occasional complication in the course of tuberculin treatment which often takes infinite care and patience to overcome. By hypersusceptibility we do not mean the usual susceptibility to an increased dose which shows itself by an ordinary reaction, but an exaggerated and growing degree of sensitiveness to the toxin which often follows an ordinary reaction without any previous warning. For instance, a patient having reacted in the usual way to 1 mg., and all the symptoms of the reaction having disappeared and the temperature become normal for two days, an injection of 0.5 mg. is followed by a marked, even a more marked, disturbance than the one produced by the 1 mg. dose. Another interval of rest being given and the dose reduced to 0.1 mg., another strong fever reaction manifests itself, and this continued and sometimes increasing susceptibility of the organism to a decreasing dose of toxin constitutes tuberculin hypersusceptibility. Years ago I was accustomed to discontinue the treatment entirely in such cases, but it is not necessary to do so. All injections should be discontinued for ten days or two weeks and then resumed again, beginning far down the scale and increasing very gradually, according to the usual schedule. Thus, if 1 mg. has been the offending dose, go back to the $\frac{1}{100}$ mg. and follow the usual schedule of increase until the patient is carried safely by the dose to which he showed such peculiar and pronounced intolerance. By the clinical method, as briefly outlined, if time enough be allowed, tuberculin immunization may be carried often to full doses: 1 c.c. of old tuberculin, of B. F. or B. E., liquid measure, with safety to the patient, and often with a marked beneficial influence on his disease.

The two most important factors in obtaining results are the length of time over which the treatment is extended and the dose of toxin the patient can be made to tolerate. Whatever reparative changes in the lesions are brought about by the treatment are necessarily very slowly produced, and it would seem that any antitoxic or antibacterial immunity obtained is also slowly induced; its degree will necessarily depend to a great extent on the amount of tuberculin to which tolerance can be established. Not much permanent good can be expected from a short treatment extending over a few months, especially if a fraction of a milligram is the highest dose reached at the time the treatment is discontinued. On the other hand, when full doses have been reached they should not be continued indefinitely for fear of exhausting the patient's power to respond, by the formation of antibodies, to the stimulus of the injection of toxin. It is well known that the horse's power to produce antitoxin can thus be exhausted, and when full doses can be reached, say 1 c.c. of the B. F., the O. T., or the B. E., no matter how long it may have taken, it is better to discontinue treatment for from three to six months and resume it again if anything in the patient's symptoms or condition seems to make it necessary.

In applying the tuberculin treatment in sanatoria the time required in most cases to attain the best results and to carry the patient to full doses is a serious objection, but it can be overcome by arranging with the patient's physician for a continuance of the injections at the patient's home, or for the application of a secondary course of injections after several months' rest when this is deemed advisable. This plan has been carried out already for some of the sanitarium patients by Dr. J. A. Miller. Whether tuberculin immunization is a strong enough influence for good to make it worth while to treat with it incipient and favorable chronic advanced cases in cities and without change in their mode of life, would be a most interesting study. I understand Dr. J. A. Miller, in New York, and Dr. Hawes, in Boston, are making a trial of the treatment under such conditions.

As to the type of cases suitable for tuberculin treatment, Denys and some of the Germans claim that even in acute cases good results may be occasionally expected by a careful course of injections, but my experience has been, with a very few exceptions, in treating patients who were apyretic or nearly so, that is, whose temperature rarely rose above 99.5° to 100°. The more chronic the type of disease the better adapted the case has seemed to me to tuberculin treatment, and most cases of a chronic type, whether incipient or of long standing and advanced, provided the nutrition is good and no serious complications exist, will derive more or less benefit from tuberculin immunization. The benefit derived may be from mere temporary improvement in all the symptoms to apparent restoration to health, with disappearance of bacilli often lasting for years. All acute cases, early cases in which the onset is acute, or advanced cases with active symptoms, should be treated by the rest and open-air method only. If they progress to cure by this method it is unnecessary to use tuberculin, and indeed I have formed the impression that it is better not to use it during the periods of activity, or at least until a partial arrest in the activity of the infection manifests itself. When the physician becomes satisfied that the patient is no longer making any improvement while treated by the open-air method, and that the case has become one of chronic phthisis, if the general nutrition is good, tuberculin injections offer a prospect of further improvement. All acute cases, cases with long-continued increasing fever, cases with very extensive lesions and who are very feeble and badly nourished, cases with intestinal and other complications, and patients who show pronounced and uncontrollable intolerance to tuberculin, are manifestly unsuitable for treatment.

The more familiar one becomes with the varying course of chronic tuberculosis, the easier it is to realize the difficulty of setting forth any positive evidence as to the favorable influence *per se* of any specific treatment when so many other factors influence the course of a disease, in itself so erratic and varying in its manifestations. The effect of tuberculin treatment on the patient is so gradual, and so slight are the apparent results, even for many months, that it is difficult to determine whether anything has been accomplished or not.

My own favorable impression of the influence of tuberculin has been principally formed by noticing how rarely the disease seemed to progress by the usual exacerbations and relapses in patients who were tolerating progressively increasing doses of tuberculin well, and in watching chronic cases who were running a slow but steadily downward course, in spite of the climatic and open-air treatment, derive marked benefit and even gradually return to apparent health after a full course of injections. It is especially in cases in which no marked intolerance to the injections has shown itself that one observes all the evidences of the chronic toxemia from which the patient suffers slowly disappear. The appetite, digestion, and nutrition all improve, the strength improves markedly, the slight accessions of temperature to 99° or 99.5° which may have been present, perhaps for years, from time to time, grow less and the temperature becomes practically normal. The subnormal morning temperature is less marked, the cough and expectoration which at certain periods in the treatment may have been increased gradually diminish, and in some cases entirely cease. In others, however, the patient may recover practically normal health, while the cough and expectoration persist apparently indefinitely, though much lessened. No marked influence on the bacilli is noted for many months in most cases. After local reactions the bacilli may greatly increase in numbers, to again decrease and occasionally disappear before the end of the treatment; but in many cases the bacilli may persist in the scanty sputum, though the patient may have regained apparent good health. In such patients, after an interval of rest, the repetition of the treatment is indicated, and a secondary course of injections is generally so well tolerated that the patient can be carried to full doses in three to four months. often without reactions.

Roemisch, of Arosa, has observed several such cases, in which after a short course of injections the bacilli entirely disappeared, and failed to reappear, though some sputum remained, which was examined negatively at intervals for as long a time as three years.

My belief in tuberculin immunization as favorably influencing the course of chronic tuberculosis rests on no more stable foundation than a strong clinical impression gained many years ago—an impression which has gradually become a conviction through years of observation. I have no statistics to present beyond what the postdischarge mortality of the patients at the Sanitarium has shown: namely, that from 18 to 25 per cent. more of treated than of untreated cases discharged from the Sanitarium during the past fifteen years were living at the time we made the inquiry. A continued study of the postdischarge mortality, extending over sufficient cases and sufficient years to exclude sources of error from other influences, can alone in time furnish unquestionable evidence as to the exact value, if any, of tuberculin immunization in prolonging life. Sahli has aptly compared its influence in the treatment of tuberculosis to that of digitalis in the treatment of heart disease, while such an excellent authority as Meissen, in Germany, denies that it has any curative influence.

That tuberculin is not the vaunted and long-looked-for specific it was at first thought to be has been amply demonstrated by the bitter experience of the past. We have learned the dangers of tuberculin treatment and its evident limitations. We have, however, also in late years learned something about the complex defensive resources of the living organism which tend to the production of immunity, and how to call them into action, though we are evidently as yet only on the threshold of the knowledge of immunization by vaccines in the treatment of chronic infections. Everything we know, however, points to immunization as the goal toward which our efforts should be directed. We have much to learn about tuberculin treatment, but even in the present state of our knowledge I am inclined to think that the production of tuberculin immunity by the mild clinical method is capable of favorably influencing the course of chronic tuberculosis, of prolonging life, and in many cases of aborting a commencing infection or extinguishing the smouldering fires of a chronic infection.

CHRONIC POLYCYTHEMIA AND CYANOSIS WITH ENLARGED SPLEEN (VAQUEZ'S DISEASE).[1]

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The syndrome chronic polycythemia and cyanosis with enlargement of the spleen is held by most writers to be a distinct clinical entity. This symptom-complex was first described by Vaquez,[2] who published a brief paper on the subject as early as 1892. It was discovered independently by Rendu and Widal[3] in association with primary tuberculosis of the spleen. More recently rather exhaustive studies of the subject have been undertaken by various observers and clinicians, more particularly Osler,[4] Türk,[5] Reckzeh,[6] Weber and Taylor, and others; and whilst these have somewhat increased our knowledge of the syndrome, quite divergent views are entertained relative to its nature and pathogenesis.

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[1] Read at a meeting of the College of Physicians of Philadelphia, April 3, 1907, and at a meeting of the Academy of Medicine, Cleveland, Ohio, March 15, 1907.

[2] Compt.-rend. Soc. de biol., 1892; Bull. Soc. des hôpitaux, 1899, p. 579.

It should be understood at the outset that polycythemia, due to a deficiency in the plasma of the blood, a common condition attributable to a great variety of definite, ascertainable causes, such as acute diarrhœa, dysentery, cholera, and the like, should not be confused with true polycythemia with enlarged spleen of obscure origin. The abnormal increase in erythrocytes of congenital heart disease, valvular affections, adhesive pericarditis, obesity, a paralyzed extremity, and of high altitudes, in the opinion of most writers, likewise differ from the form under consideration. The hyperglobulism observed in ether anesthesia and phosphorus poisoning should also be excluded.

Etiology and Pathogenesis. At present writing it is believed by certain observers that stagnation of the blood due to a variety of factors is the sole cause of the symptom-complex. For example, in a case reported by Reckzeh, occurring in a young man, a malignant tumor of the thymus gland and lung caused gradual compression of the superior vena cava followed by polycythemia (limited, however, to that portion of the body which was affected by the venous obstruction), chronic cyanosis, and splenic enlargement. The lesson taught by the case in question was confirmed by experiments on two animals to the satisfaction of Reckzeh; he found that compression of the superior vena cava caused sufficient stagnation of the blood current to induce the development of a moderate polycythemia (6,200,000).

A. N. Hall[7] suggests that the polycythemia may be a compensatory process—an attempt to make up the deficiency in oxygen-carrying capacity of the individual cells by an increase in the number of cells. Moreover, polycythemia and enlargement of the spleen has been observed in obstructive cardiac lesions. Thus E. Weil[8] found red transformation of the bone-marrow (the normal fatty tissue having disappeared) in postmortem examinations on two children with congenital pulmonary stenosis and chronic cyanosis. As stated above, most writers contend that polycythemia and enlarged spleen is a condition which is not synonymous with chronic cyanosis due to chronic valvulitis.

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[3] Bull. et mém. Soc. des hôpitaux, 1899, III Ser., p. 528

[4] Trans. Assoc. Amer. Phys., 1903, p. 316

[5] Wien. klin. Woch., 1904, Nr. 6 u. 7.

[6] Ztschr. f. klin. Med., 1905, lvii, 215.

[7] Amer. Med., June 27, 1903.

[8] Compt.-rend. Soc. de biol., 1901.

It can be readily understood how an unequal distribution can show polycythemia on examining the peripheral blood. It is a recognized fact that local cyanosis from any cause calls forth an increase in the number of red cells. It would appear, therefore, that any condition accompanied with cyanosis should be capable of inducing polycythemia.

On the other hand, it has been claimed that polyglobulism is ascribable to an overproduction of erythrocytes as a primary change, and this view is supported by the necropsy findings of a case reported by Weber and Watson:[9] they had convincing evidence that an increased production of erythrocytes was taking place in the bone-marrow. These observers, then, are inclined to regard the changes in the bone-marrow as primary, or, if not strictly speaking primary, as representing an excessive "vital reaction" to stimulating agents.

Weber and Watson account for the symptom-complex of the condition on the supposition that the osmotic tension of the blood of patients with extreme polycythemia is higher than that of ordinary blood. Bence[10] holds the syndrome to be primarily due to an overproduction of red cells; he argues that the qualitative change in the various anatomical elements of the blood point to increased erythroblastic activity of the bone-marrow. Widal, assuming tuberculosis of the spleen to be a uniform pathological finding, thinks the diminished function of that organ produces increased activity of the bone-marrow.

Türk believes it to be a primary affection of the bone-marrow. Rosengart[11] found evidence of hyperplasia of the leukocytic myeloid tissue, including mast cells, myelocytes, and erythroblasts, in the spleen and liver. In one case this observation led Türk[12] to suggest that the syndrome was dependent on hyperplasia of this erythroblastic myeloid tissue plus the histological changes noted in the liver and spleen by Rosengart, a condition analogous to the hyperplasia of leukocytic myeloid tissue in myelogenous leukemia.

These changes, however, have not been noted by other observers. A. V. Korauspi[13] argues that the deficiency of carbon dioxide in the blood demands an increased hemoglobin content, hence also a polycythemia to maintain the normal acidity of this tissue. Limbrea first pointed out that polyglobulism goes hand in hand with carbon monoxide poisoning. Julius Bence found experimentally that the inhalation of carbon monoxide decreased the erythrocyte count from 9,860,000 to 8,000,000 and a simultaneous, distinct falling off in the viscosity was noted. He regards the fact that the inhalation of carbon monoxide produces a rapid reduction of the erythrocytes in these cases as prima facie evidence that the polycythemia is the result of a diminished percentage of this gas in the circulating blood.

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[9] International Clinics, 1905, vol. iv, 14th series, p. 47.

[10] Deut. med. Woch., 1906, xxxii, Nr. 37.

[11] Mitteil. aus den Grenzgeb. der Med. und Chir., 1903, iv, 495.

[12] Wien. med. Woch., 1902, Nr. 5, p. 226

[13] Quoted by Bence, Deut. med. Woch., September 13, 1906.

Among the earlier reports of cases, principally by French writers, tuberculosis of the spleen was noted. But cases of splenic tuberculosis have been known to occur without polycythemia and cyanosis. For example, Reckzeh reports a fatal instance of tuberculosis of this organ, as shown clearly by an autopsy in which the characteristic features of polycythemia and cyanosis failed to appear during the life of the patient. Türk also reports two of his own cases, and four additional cases from the literature, in which tuberculosis of the spleen was wanting. It is quite certain that whilst the association of splenic tuberculosis with polycythemia and cyanosis is sometimes met with, the latter may occur without he presence of the former.

My observations in Case I, reported below, leads me to the conviction that defective venous tonus plays an important role in the pathogenesis of the condition. The nervous phenomena are at times, at least, well-marked and peculiarly distressing, as shown by the history of one of the subjoined cases.

Case I.—A. W., female, single, aged twenty-five years; height 5 feet 6 inches; weight 198½ pounds; was referred to me by Dr. Finney, of Baltimore. First came under observation September 23, 1904. The family history showed Bright's disease among the mother's antecedents; also eczema, rheumatism, and cases of moderate obesity. The mother, however, is living and in vigorous health; the father is deceased, of organic heart disease complicated with chronic nephritis, at the age of forty-two years. One brother was a hemophiliac; otherwise the family history was unimportant.

The patient had not contracted any of the usual childish diseases except measles at twelve years of age, at which time she arrived at puberty. Later she suffered from colds in the head, with much catarrhal discharge. At seventeen years of age she had congestion of the lungs, and the eyes had been bad for fifteen years. From the eleventh to the twenty-third year of age the patient experienced one or two attacks of nose-bleed each month. Menstruation was formerly painful, but not now; it is irregular, however, recurring every two or three months, and consisting of a dark-brown discharge. Patient has pursued active physical exercise and has dieted with a view to reducing the bodily weight, but without results. Cyanosis, which had slowly increased, was first observed ten years since.

Miss W. has been inclined to rheumatism for eight years, and subject to severe headaches during the same period. For one year prior to her first visit (September 23, 1904) the headaches had improved, but the cyanosis had become worse; at the same time she also suffered from violent cramps in the legs and feet. At the date of her visit the mucous surfaces were somewhat less livid than the skin. For several years the patient has been timid, mentally distressed, and apprehensive to a degree. She complained of slight vertigo at intervals. The urine analysis proved negative, as a rule, in its results. On one occasion, however, a reaction for sugar was obtained, the percentage being less than one.

A blood examination on four different occasions gave the following average result: red cells ranged from 5,300,000 to 6,960,000. The hemoglobin value averaged 110 per cent., while the leukocytes varied from 13,600 to 20,000. The red cells are overburdened with hemoglobin; they stain about normally and are of natural size and form. The white cells show mostly polymorphonuclear elements.

Physical Examination. The patient is a well-developed female subject, with good musculature and prominent panniculus adiposus. The face is markedly cyanotic, presenting a livid-red appearance everywhere except around the mouth, chin, base of the ears, and a narrow border around both eyes, the latter areas looking blanched by contrast. Both cheeks and forehead show a far greater degree of cyanosis than the rubicund face of plethoric obesity. As in other cases reported, pressure upon the skin left a decided anemia, which slowly became effaced. Examination of the superficial veins of the face conveyed the impression that they were greatly lacking in tonicity. The apex beat is indefinable, owing to corpulency. An examination of the lungs proved negative in its results. The heart shows slight enlargement of the left ventricle. It is difficult to delimit the organ, however, owing to the subcutaneous fat deposits. The first sound is somewhat muffled, the second aortic slightly accentuated, and the action of the heart is abnormally rapid, the pulse ranging from 110 to 120. There is no evidence of arteriosclerosis and the blood pressure tested by the Riva Rocci blood-pressure instrument—systolic 145, diastolic 115. The spleen is found to be slightly enlarged. The liver is of normal dimensions; also the thyroid gland. No other abnormal physical signs are discoverable.

The treatment was directed toward the reëstablishment of the menstrual function, and after eighteen months the menses occurred every six weeks. The patient has been seen at intervals of two or three months from the date of my first examination until the present, and the course of the complaint has been marked by recurring exacerbations and remissions. The headaches, however, have been practically absent for a year or longer, or since the menses have been more nearly normal; the distressing feeling of apprehension has also undergone noticeable improvement, particularly during the last six months, and the same is true of the cyanosis. At date of writing (January 20, 1907) the nervous tremor, mental apprehension, and the endurance show marked improvement. It is worthy of note that the nitrites afforded considerable temporary relief from the headaches from the beginning, thus showing that the cause of the headaches most probably was in part at least heightened tension in the cerebral vessels.

Case II.—A male, aged thirty-one years, a clerk; height 5 feet 9 inches; weight 125 pounds; came under my care June 7, 1904. The mother died of childbirth at forty-five years of age. The father is living and healthy. He has brothers and sisters, all of whom enjoy good health. The previous diseases from which the patient suffered were mumps, whooping-cough, and two attacks of influenza in adult life; he has also been susceptible to "colds" in the head and chest, which he ascribes to catarrh of the nasopharynx.

The social history shows moderate indulgence in tobacco and alcohol, the free use of coffee, rarely tea, and the usual indifference to diet. There is no history of either gonorrhœa or syphilis. The patient is married and has three children, all healthy. He has complained of indigestion at intervals for the past fifteen years, the principal symptoms being fulness, eructation of gas, and discomfort in epigastrium, shortly after the ingestion of food; the bowels act regularly; the vision is blurred at times, and headaches, mental apprehension, and slight dizziness have been the chief nervous phenomena; for several years the patient has become easily fatigued on muscular exertion (asthenia); the legs are weak and also the grasp; he sleeps well; during the day he has considerable cough attended by mucoid expectoration and thoracic pains, which radiate to the lumbar regions.

The *physical examination* shows at a glance cyanotic lips, conjunctivas, ears and face (especially the nose); the hands and feet are also quite dusky. This cyanosis had been present for five years. The left ventricle is somewhat enlarged (hypertrophied), a faint systolic murmur is heard at the apex, and the second pulmonary sound is moderately accentuated. The spleen is palpable; but not markedly enlarged. The pulse is somewhat accelerated, but the tension is not abnormally high. The urine contained no albumin. A blood examination by Dr. Batroff showed: red cells, 7,400,000; leukocytes, 12,600; hemoglobin, 130 per cent.; color index 0.88 per cent. The differential count gave this result: polymorphonuclear leukocytes, 69.30; small lymphocytes, 23.72; large lymphocytes, 6.5; eosinophiles, 0.5 per cent.; absence of poikilocytes, macrocytes, microcytes, and nucleated red cells. The red cells were of uniform diameter, and contour was perfectly preserved; no crenation or other abnormality, and the staining capacity exceedingly good.

The patient has been seen at long intervals until the present time. The principal complaint at these visits was nervous apprehension, momentary dizziness, and disinclination for exertion, either mental or physical. As the result of treatment, chiefly dietetic, the digestive function has been improved, but the cyanosis and nervous phenomena have been only slightly relieved, if at all. Small doses of cardiac stimulants, such as digitalis and strychnine, seemed to afford relief from the cyanosis and increase the general strength, but only temporarily. The nitrites also failed to bring about lasting improvement, although affording some degree of relief from the cerebral symptoms.

Case III.—A third case, in which the diagnosis may be questioned on some accounts, came under my notice September 23, 1904, through the kindness of Dr. Prickitt, of Mt. Holly. The history is as follows:

E. A., aged twenty-one years; single; height 5 feet 3 inches; weight 86 pounds. The father is deceased, from necrosis of the leg following an operation; the mother lives and is healthy; has two brothers and two sisters who enjoy good health. Apart from neuropathic heredity on the mother's side, the family history is of no etiological importance.

Patient experienced the usual childish diseases, and at twelve years of age developed chills, accompanied with fever and sweats, (presumable malaria) lasting several weeks. Later, or when seventeen years of age, was operated on for appendicitis (primary attack); at the age of nineteen years received a blow by a hard ball over the right ovary. This was followed by inflammation and three abscesses which ruptured into the uterus; cystitis followed, and two years later, May, 1905, curettement was performed; subsequently acute nephritis developed. The menstruation began at eleven years, has been somewhat painful, and since the operation for appendicitis has shown a markedly irregular tendency.

The present illness dates from the operation for appendicitis, four years ago, although no cyanosis developed until after the curettement in May, 1905. The urine is scanty, dark, alkaline in reaction, and slightly albuminous; no casts are present. Physical exertion increases the cyanosis and induces dyspnœa. The patient is weak and neurasthenic.

The skin is markedly livid, especially that of the face, and there is obvious inspiratory dyspnœa; the pulse is rapid, small, and compressible, and the heart slightly dilated, and an occasional systolic murmur is audible at the tricuspid orifice. The abdominal walls are tense and hard to the feel; no splenic enlargement is detectable, and there is no œdema of the lower extremities.

The blood examination by L. N. Boston shows a cyanosed condition of this tissue; blood flow from puncture free; hemoglobin, 120 per cent.; erythrocytes, 4,290,000; leukocytes, 11,100. The stained specimen reveals many poikilocytes, a few macrocytes, and some nucleated red cells; all cells overstained and very deeply. A subsequent blood examination (made by W. C. Batroff) gave a somewhat different result: the blood flows freely from puncture, and resembles chocolate in appearance; the hemoglobin value from a cyanosed finger is 79 per cent., from a cyanosed ear 110 per cent.; red cells 4,800,000, white cells 8400; differential count of leukocytes shows: polymorphonuclear 67 per cent., small lymphocytes 24 per cent., large lymphocytes 7 per cent., eosinophiles 1 per cent., mast cells 1 per cent. The corpuscles are generally large, with decoloration of the centres; a few poikilocytes, few macrocytes, but no erythroblasts were found.

In this case the structure of the blood is somewhat abnormal, and while the color index shows no anemia present, but rather the reverse, the red-cell count (4,290,000 to 4,800,000) is comparatively low, and there are abnormalities in form and size of the corpuscles. It scarcely seems tenable to suppose that the principal difficulty in this case consists in deficient oxidation, owing to the absence of an increase of the red corpuscles, to account for the excessive hemoglobin content of the erythrocytes. A tabular record of my own and all the cases not included in Reckzeh's list is herewith appended:

TABLE I.

Case	Author	Sex, age	Splenic enlarg.	Albuminuria.	Hemorrhage.	Result	Remarks
1	Weber	F. 37	Yes	No	No	Improved	Absence of cyanosis
2	Zimlick	M. 32	...	No	No	Died	
3	Collins	F. 24	...	No	No		
4	Hall	F. 61	No	0.05%			
5	"	F. 40	No				
6	Weber and Watson	M. 58	Yes	0.5%	Hematemesis (?) 5 yrs. ago	Died	Cause of death, syncope
7	Engelbach and Brown	F. 40	Yes	Trace	No	No change	Duration 11 yrs.
8	Bence	M. 43	Yes	1%	No	"	No apparent obstruction after 5 yrs., but 9 yrs. cyanosis
9	"	F. 45	Yes	No	....	"	Short period. Began 2 yrs. ago
10	"	F. 42	Yes	No	....	"	Began 2 yrs. ago from gums bleeding
11	Koranyi[14]						
12	Hutchinson and Miller	M. 45	Yes	Trace	Yes	Died	Died of coma and hyperpyrexia. Duration 7 yrs. 2 mos.
13	Begg and Bullmore	F. 47	Yes	No	....	Improved	
14	Lommel	M. 42	Yes				
15	Robertson	M. 48	Yes	0.7%	Yes	....	Obstinate constipation
16	Aldrich and Crummer	F. 53	Yes	No	No		
17	Reissmann	F. 18	No	...	Yes	....	Signs of venous obstruction in chest and legs
18	Anders	F. 25	Yes	None	Yes	Improved	
19	"	M. 31	Yes	...	....	....	Began 10 yrs. ago

Remarks. My own figures coupled with those of Reckzeh (not included in my tables) give a grand total of 53 cases. These will form the subject of a few observations and inferences.

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[14] No details. Case referred to by Dr. Bence. Treated with oxygen inhalations.

TABLE II.—BLOOD EXAMINATIONS.

Author.	Erythrocytes.	Size and form.	Normoblasts.	Polychromatophiles.	Leukocytes.	Lymphocytes.	Leukocytes, mononuclear and transitional.	Polymorphonuclear.	Eosinophiles.	Mast cells.	Myelocytes.	Hemoglobin. %
Bence, Case I	10,340,000 9,440,000	Practically normal	1 1	None "	12,500 6,400	9.2 12.3	1.7 mono. ..	80.0 78.6	6.7 4.6	3.1	...	180 (Gowers)
" " II	8,000,000	....	..	..	....	....	..	....	....	....	...	27.2
" " III	8,350,000	....	..	.	8,400	....	..	....	....	....	...	27.8
Engelbach & Brown	10,909,000 8,037,000 12,584,000	....	.. .. ..	.. Few	9,600 (9 to 11%) 6,600 9,600	.... 11.0 to 14.0	5.0 mono. 6.0 trans.	.... 75.0 to 76.0	.... 1.0 to 1.5	.... not increased	...	above 120
Zimlich	6,480,000 9,240,000	....	.. ..	.. ..	13,200	12.0	5.0	85.0	2.5	....	...	120+ 120+
Ascoli	?	....	..	..	15,000	16.0	11.0	51.0	20.0	2.0	...	95, was 110
Weber	8,240,000 10,960,000	....	.. ..	.. ..	4,000 9,000	17.25	8.5	66.8 81.0	0.5	1.0	...	120 185
Hall, Case I	9,949,600	Normal	..	..	6,500	....	..	....	....	....	...	170 200
" " II	9,935,000	....	..	..	22,000	....	..	....	....	....	...	160-170
Collins	9,821,000	Normal	..	..	17,800	26.0	..	73.0	0.5	....	None	110
Weber & Watson	10,000,000 11,000,000	Practically normal	.. ..	.. ..	7,500 8,000	17.4	..	82.4	0.2	....	None	170
Koranyi (quoted by Bence)	7,400,000											
Hutchinson & Miller	8,000,000 11,000,000	Nothing abnormal	.. ..	.. ..	 17,160	5.6	2.4	82.8	2.8	2.4	...	110 120
Begg & Bullmore	6,850,000	....	..	..	11,300	13.8	4.0	77.3	1.5	....	...	100
Lommell	8,230,000	....	..	None	11,000							
Aldrich & Crummer	7,700,000 6,048,000	Variation in size and contour	.. ..	.. ..	4,700 5,500	19	..	72.5	4.0	....	4.5	120 90
Anders, Case I	5,300,000 5,960,000	Natural, overburdened with hemoglobin	.. ..	.. ..	22,000 1,320,000	38.05	.	61.9	0.5	....	...	110 110
" " II	7,400,000	Normal	..	..	12,600	29.77	..	69.3	0.5	....	...	130 color index 0.88%

Sex. According to most writers, males are more prone to the affection than females, but of the 18 cases that I have collected, in which the sex is given, 11 (or 61 per cent.) were in females, and 7 (or 39 per cent.) were in males. Engelbach and Brown state that an "analysis of all the cases shows it to be about equally divided between the sexes."

Race. Of 7 cases reported by Türk, 5 occurred in Hebrews, and racial predisposition is generally conceded. Precise figures to indicate the influence of race, however, are wanting.

Age. According to Reckzeh, the great majority of the cases occur in the middle period of life. On the other hand, Türk's figures (based on a total of 20 cases) show that it occurs principally in adults. In my group of 18 instances the age was recorded, and of these 3 occurred between twenty and thirty, 3 between thirty and forty, 9 between forty and fifty, and only 3 after fifty years of age. My results, therefore, are in consonance with Reckzeh's, rather than with those of Türk.

Spleen. Reckzeh has pointed out that in only 2 (Osler, Cabot) out of 34 cases was there an absence of splenic enlargement. Of the 18 cases recorded in my table, splenic tumor occurred in 14, or 78 per cent. It was present in 44 out of a total of 49 cases collected from various sources by me, or in 89.8 per cent.

Albuminuria. This was present in 43 per cent. of the cases in my series in which the symptom had been investigated. Engelbach and Brown state that albuminuria occurs in one-half of the cases, in some in large amounts.

The most characteristic blood-finding is polycythemia. It has been held by certain writers that an erythrocyte count of less than 8,000,000 does not constitute polycythemia with enlargement of the spleen. On the other hand, Cabot holds that the red cells number about 7,000,000 in a typical case. Unquestionably, cases which presented every other characteristic clinical feature have been reported in which the erythrocyte count was below these figures. In Case III of my series (not included in my table) the erythrocyte count was only slightly above normal, if we regard 4,500,000 as the normal standard in the female. In the majority of instances, however, the red cells number over 8,000,000, as may be seen from a glance at Table II.

In most cases of polycythemia a moderate leukocytosis is present, although a few have been reported in which a leukopenia was found.

Cases have been reported in which the leukocyte count fell short of that of the erythrocytes. Thus in a case recorded by Osler only 4000 leukocytes per c.mm. were noted, while the red cells numbered 9,953,000; hemoglobin 120 per cent. On the other hand, we see in certain reported cases an increase of the leukocytes out of ratio to the erythrocyte count. For example, in some cases in which the red cells did not exceed 7,000,000, the white cells amounted to over 20,000. The cases just related belong to this category. I mention these facts with a view to emphasizing the point that mere obstruction to the circulation, or blood stasis, does not account for this symptomatic manifestation.

Of 18 cases in which the leukocyte count is given in my table, 14 showed an increased number of leukocytes. Attention has been directed to a disproportionate increase in the polymorphonuclear leukocytes as an evidence of primary disturbance of the blood-forming organs. This finding, however, did not obtain in my 2 cases, and the table indicates the following in this respect: 7 cases in which the polymorphonuclear elements were relatively increased, and 7 cases giving a ratio at or below the normal. It would seem that the normal proportion of the different forms of white cells is maintained, as shown by the differential count, in about one-half of the cases at least. Analyzed with regard to the hemoglobin percentage, an examination of Table II will show that of 16 cases in which this percentage is reported 10 are above 120 per cent., 1 shows only 90 per cent., and another 100 per cent. In 1 of Hall's cases the hemoglobin value was 200 per cent.

Is the abnormal blood-forming power a physiological or a pathological process? It is clear that on account of the increase in the value of the various elements of the blood, which increase is usually more or less proportional, the blood-generating organs are stimulated, but the question arises, Is this a primary or a secondary role? Vaquez was the first to suggest that primary tuberculosis of the spleen was the cause of the syndrome. Bence, Widal, Türk, Osler, Rosengart, and Weber and Watson, previously named, all contend for a form of polycythemia with splenic enlargement, either of unknown etiology (Osler) or due to splenic tuberculosis or to a primary overproduction of erythrocytes from some stimulating agency not as yet discovered. As pointed out by Engelbach and Brown, and others, however, many cases have more recently come to autopsy, in which neither the spleen nor any other organs showed tuberculous lesions; hence this morbid process cannot be held responsible for all cases. In my cases and in many reported by other observers the blood examination failed to show changes in this tissue of sufficient importance to justify the inference that the blood-making organs are directly affected. W. Egbert Robertson informs me that in his case (Table I) no primary disease of the hematopoietic organs was indicated by the microscopic findings, the blood having been, on one occasion, taken from the spleen notwithstanding.

At present the balance of testimony is clearly in favor of the view that in many cases described as polycythemia and cyanosis with splenic enlargement, the extraordinary blood-making power, as shown by the increased number of red cells in the bone-marrow, is probably secondary or physiological and quite analogous to what is sometimes seen after excessive hemorrhage.

In this connection certain well-established effects of venous hyperemia, due to some form of obstruction, should be briefly presented. In the first place, the velocity of the blood current is decreased in stasis, and although more than the usual number of corpuscles occupy the capillaries and veins, the temperature is low as compared with that of a part or parts of the body the seat of arterial congestion. As venous congestion advances with slow increase in the size of the veins and capillaries, the metabolic processes are profoundly affected and lowered, rendering the tissues abnormally susceptible to the influence of microbic and all other forms of irritants.

The cyanotic induration of the various viscera (liver, spleen, kidney) in consequence of continuous venous hyperemia is well known. Here the supporting structure of the organs is strengthened by the formation of fibrous tissue. Now, it is reasonable to suppose that analogous changes occur in the nervous structures, parts of the brain, spinal cord, ganglia, and nerve shafts that are endowed with connective tissue. We may assume that all tissues which are the seat of prolonged venous engorgement present pathological changes which are far-reaching and variable in character. The query here arises, Can we ascribe the nervous phenomena observed in chronic polycythemia and cyanosis with enlarged spleen to the effects of the venous obstruction? It is a matter of common observation that in chronic valvulitis the principal nervous features arise after failure of compenstion, or at a time when engorgement of the veins is present. When the left ventricle fails to maintain the movement of the blood in the capillaries (*e. g.*, in aortic incompetency), insomnia, headache, vertigo, restlessness, and weariness are observed. Again, emotion may induce palpitation. These symptoms all have their parallel in cases of chronic polycythemia with cyanosis, and the same is even more true of that prominent symptom, dyspnœa on muscular exertion. The comparison also holds, although less completely, with reference to the general neurasthenic symptoms.

In cases of true polycythemia and cyanosis with enlarged spleen there is a painful nervous apprehension, such as is witnessed in functional diseases of the heart arising from conditions outside of this organ. Every clinician of wide experience, however, must have met with structural affections of the heart in which the nervous equilibrium is more or less disturbed by this class of phenomena, *e. g.*, when the heart muscle is hyperesthetic from exhaustion.

The symptomatology of the condition, so far as observed, may be accounted for, in great part at least, by the persistent cyanosis, and the polycythemia may be looked upon as a result of the venous stasis; and this hyperglobulism is intended to compensate for the diminished oxygen-carrying capacity of the blood.

As stated above, however, true polycythemia and cyanosis with enlargement of the spleen, according to the majority of writers, exhibits a wider range of symptoms, as a rule, the nervous phenomena being especially conspicuous in the clinical picture, and the blood findings recorded in some cases, it has been justly claimed, are at variance with those found in mere stagnation.

It may be argued that if stagnation alone were the cause, the syndrome would probably show greater prevalence than it has done. Again, the cases would naturally subdivide themselves into local and general polycythemia according to the character and location of the vessels. I regard it as probable that there is a form of primary polycythemia of unknown etiology, characterized principally by marked polyglobulism and other hemic features, cyanosis, headache, vertigo, and splenic enlargement; but it must be of rare occurrence, while it is clear that the majority of the cases which have been reported and classed as instances of this condition have had a different pathological etiology, although closely simulating the primary variety clinically. Finally further proof, both clinical and pathological, is required before any of the theories thus far advanced can be accepted.

Treatment. As regards treatment, it may be said that no special methods thus far advanced have been accepted as generally applicable. The environment is to be made as favorable as possible physically and mentally. A light, nutritious diet, an abundance of fresh air and gentle methodical exercise, should be advised and encouraged. Of medicines, the nitrites have been most extensively employed, and they have afforded relief to the headache and other cerebral symptoms in some cases at least. As stated by Watson, venesection with or without the injection of saline, has been followed only by temporary effects. Begg met with a case, presumably of malarial origin, in which the vigorous treatment adopted in the East in such instances was resorted to, viz., the tri-iodide of mercury ointment was applied over the spleen, and the part exposed to artificial heat, while quinine was given in increasing doses up to 45 grains a day. As the result of this treatment, the few pathological elements shown by the blood examination progressively diminished; the spleen, which was enlarged, retracted upward two inches in the mammary line. That the principal benefit in Begg's case arose from the massive doses of quinine was shown by the fact that the temporary discontinuance of the remedy was followed by an increase in the size of the spleen. The results obtained in this case also point to a possible malarial origin of chronic polycythemia and cyanosis with enlarged spleen, in some instances at least. If this view be accepted, it is obvious that the blood changes presented by the syndrome in such cases are to be classified as secondary.

The viscosity of the blood is greatly increased in this condition, as shown by Weber and Watson; hence, on rational grounds, the use of iodides should be advised. In my cases, however, their exhibition was without obvious beneficial effects. In Case I of my series, in which amenorrhœa and moderate obesity were present, improvement was noted on the reëstablishment of the function of menstruation.

THE PROGNOSIS OF TRANSIENT SPONTANEOUS GLYCOSURIA, AND ITS RELATION TO ALIMENTARY GLYCOSURIA.

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There are few diseases of metabolism about whose beginning less is known than that of diabetes, and the gratifying results so often obtained from prompt and intelligent treatment, even in advanced stages, make this obscurity of inception the more regrettable. We possess definite knowledge of the commencement of that very small group of diabetic cases following traumatism or disease of the nervous system, and of that group following or accompanying obesity, but of the early stages of the ordinary form, embracing the great majority of cases, we know very little. The few reported cases which have been followed from the beginning have shown at first either spontaneous glycosuria under ordinary normal conditions, or an alimentary glycosuria following ingestion of glucose, and later have gradually developed constant glycosuria and diabetes.

A preliminary consideration of the nomenclature will be of some help in understanding the discussion of our subject. The term transient or intermittent glycosuria is a very broad one, and includes practically all non-diabetic glycosurias, for all continuous glycosurias are diabetic. The adjectives used to describe the various glycosurias have, generally, some reference to their etiology; thus hepatogenous, that form arising from any disease or injury which causes the liver to discharge rapidly its glycogen; neurogenous, that form resulting from disease or injury of the nervous system (probably to be included under the previous variety); and alimentary, that form arising from the ingestion of carbohydrate food, etc.

Alimentary glycosuria, when arising from intake of any sugar, is termed *e saccharo;* when from starch, *ex amylo* (invariably diabetic); and when from a mixed diet, spontaneous. Spontaneous glycosuria may be transient or permanent, and if the latter is, of course, diabetic. The transient spontaneous glycosuria includes those cases in which we can rule out the other various causes of glycosuria, but of which we do not know the primary cause. The secondary cause, of course, is inability to assimilate the starch and sugar of a mixed diet, but whether ultimately this is to be attributed to some transient disturbances of metabolism or to a beginning diabetes, we do not know. The usual term for this glycosuria is simply transient or intermittent, but transient spontaneous seems a more suitable term, and we shall use it to designate those transient glycosurias of unknown origin appearing on an ordinary mixed diet. The part transient disturbances of the pancreas play in the production of many of these non-diabetic glycosurias is yet to be definitely determined. It should be borne in mind that there is a physiological alimentary glycosuria, caused by the ingestion of excessive amounts of any sugar, which has no pathological significance whatsoever.

The existence of many forms of transient non-diabetic glycosuria has led us in the past to underestimate, perhaps, the possibly serious import of an intermittently appearing spontaneous glycosuria, but of late we have been inclined to give a grave prognosis in these cases. Von Noorden[1] has recently expressed the following opinion: "When a transitory glycosuria is accidentally discovered in an individual, the reason for which does not obviously lie in a diet excessively rich in sugar, I should like to recommend urgently that such an event be neither heeded lightly nor neglected. It is in most cases the warning signal of later diabetic disease, the outbreak of which may not occur for many months or even years." No definite statistics are quoted in support of this view.

We have been able to find in the literature but 9 cases of transitory or spontaneous glycosuria which were followed until they developed diabetes. These cases are reported by Raphael,[2] Kraus and Ludwig,[3] H. Straus,[4] Bierens de Haan,[5] and Van Oordt;[6] Cantani[7] also speaks of such cases, but gives no figures. The time elapsing between the first observed glycosuria and the development of diabetes in all cases was one year or less, with the exception of von Norden's case of diabetogenous obesity, in which the time was four years.[8]

Answers to the questions, (*a*) How many of the cases of spontaneous glycosuria develop later into diabetes? and (*b*) Is there any way of identifying these cases at an early date? would add to our knowledge of this disease, enable us to give a more intelligent prognosis in these very perplexing cases, and possibly spare some patients a most irksome treatment.

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[1] Disorders of Metabolism and Nutrition: Diabetes. English trans., 1905, p. 147.

[2] Ztschr. f. klin. Med., xxxvii, p. 20.

[3] Wien. klin. Woch., 1891, p. 855–897.

[4] Deut. med. Woch., 1897, Nos. 18 and 20.

[5] Archiv f. Verdauungsk., Band iv, Heft 1, p. 4.

[6] Münch. med. Woch., 1898, No. 1.

[7] Der Diabetes Mellitus, 1877, German trans., 1880, p. 305.

[8] Verhandlungen des XIII Congress f. innere Medizin, 1895, p. 140.

Name.	Age	Sugar history.	Date of last normal urine examination.	Subsequent history.	Urine examination.	Fehling's.	Phenyl hydrazin.	Polariscope.	Fermentation.	Rotation after fermentation.	Remarks.
D. W.	34	Trace, 1900.	None made.	Healthy, gained 28 pounds.	24 hour spec., 1200 c.c., s. g. 1025.	—	—	per ct. — 0.15			
					2 hour post-glucose.	—					
					3 hour post-glucose.	—	+	— 0 3	—	......	Free from diabetes, 1906.
A. A. C.	24	0.42 per cent., 1901.	None made.	Healthy.	Before and after lunch spec.	—	—				
					2 hour post glucose spec.	—	+	— 0.2			
					3 hour post-glucose spec	—	—	— 0.225	—	— 0.2 per cent.	Free from diabetes, 1906.
D. V. W.	44	0.83 per cent., 1901.	1898 (Ins. Co.).	May, 1906, trace albumin, no sugar, hyaline and gran casts. 1901 (6 weeks later), 0.48 per cent. glucose. Accepted by two Ins. Cos. Healthy.	24 hour spec., 817 c c., s. g. 1026.	—	—				
					2 hour post-glucose.	—	—	0.0			
					3 hour post-glucose.	—	—	0 0	...	......	Free from diabetes, 1906.
H. M.	53	0.83 per cent., 1901. Trace of albumin; hyaline casts.	None made.	Lost 40 pounds. 1904, chronic nephritis and uremia. 1905, the same. 1906, chronic nephritis.	24 hour spec., 1400 c.c., s. g. 1020.	—	–	0.0	...	......	Free from diabetes, 1906.
J. N.	37	0.37 per cent., 1901.	None made.	April, 1901, no glucose, accepted by three companies since. Healthy.	13 hour spec., 915 c.c., s. g. 1022.	—	—	0.0	...	......	Free from diabetes, 1906.
P. E. G.	36	Trace, 1895. 0 83 per cent., 1901 (0.25 per cent. next day).	None made.	Healthy, urine frequently negative for sugar.	24 hour spec., 1500 c.c., s. g 1018.	—	—	0.0			
					2 hour post-glucose.	—	—	— 0.25			
					3 hour post-glucose.	—	—	— 0.5	...	......	Free from diabetes, 1906.
A. B. H.	43	Small quantity, 1900.	None made.	Healthy, urine negative once.	24 hour spec., 1550 c.c., s. g. 1015.	—	—	0.0			
					2 hour post-glucose.	—	—	— 0 05	—		
					3 hour post-glucose.	--	—	— 0.1	...	......	Free from diabetes, 1906.
A. S. S.	39	0.67 per cent., 1901.	1898.	Healthy, 2 urinalyses since negative.	12 hour spec., 850 c.c., s. g. 1027.	—	—	0.0			
					2 hour post-glucose.	—	—	0.0			
					3 hour post-glucose.	—	—	0.0	—	......	Free from diabetes, 1906.
I. A. B.	31	0.33 per cent., 1900 (found twice).	None made.	Has lost 30 pounds, extremely neurotic. Urine frequently negative. Nov., 1902, 0.66 per cent. glucose (Ins. Co.). 1903, cane-sugar test neg. (Ins. Co.).	24 hour spec., 730 c.c., s. g. 1025.	—	—	0.0			
					2 hour post-glucose.	—	—	— 0.1	—		
					3 hour post-glucose.	—	—	— 0.2	...	......	Free from diabetes, 1906.

I. O.	51	0.42 per cent., 1900.	None made.	Healthy, urine frequently negative for glucose (physician).	24 hour spec., 1100 c.c., s. g. 1020.	—	—				
					2 hour post-glucose.	+	+	— 0.5			
					3 hour post-glucose.	—	+	— 0.25	...	......	Free from diabetes, 1906.
H. T. A.	27	0.42 per cent., 1901.	1893 (Ins. Co.).	Accepted twice by Ins. Co. 2 attacks pneumonia. July, 1903, after 100 gm. cane sugar, 0.67 per cent. glucose (trace glucose 1903, after sugar meal).	24 hour spec., 860 c.c., s. g. 1020.	—	—	0.0	—		
					2 hour post-glucose.	—	—				
					3 hour post-glucose.	—	—	......	...	......	Free from diabetes, 1906.
C. K.	32	Trace, 1895, 1.1 per cent., 1901.	1890 (Ins. Co.).	1903, trace sugar (Ins. Co.). Healthy, urine frequently negative.	24 hour spec., 1020 c.c., s. g. 1030.	—	—	0.0	...	......	Family history, diabetes; very suspicious case.
					2 hour post-glucose.	+	+	+ 0.3	+		
					3 hour post-glucose.	—	—	— 0.125	—		
M. G.	33	0.56 per cent., 1901, none next day.	None made.	Accepted by 2 companies. Jan., 1902, 3.3 per cent. sugar Jan., 1904, 1.1 per cent. sugar. Healthy.	24 hour spec., 1010 c.c., s. g. 1022.	—	—	0.0			
					2 hour post-glucose.	+	+	+ 0.3	+		
					3 hour post-glucose.	—	—	— 0.125	—	......	Very suspicious case.
W. K.	41	Small quantity, 1901.	None made.	Healthy, urine frequently negative for glucose. Dec., 1902, 0.56 per cent. Dec., 1903, after 100 gm. sugar 1.2 per cent. glucose.	12 hour spec , 780 c.c., s. g. 1026.	—	—	0.0			
					12 hour spec., 265 c.c., s g. 1024.	—	—	0 0			
					2 hour post-glucose.	+	+	+ 0.1	—		
					3 hour post glucose.	—	—	— 0.1	—	......	Suspicious case.
W. B.	43	0.67 per cent., 1900.	None made.	Healthy.	12 hour spec., 635 c.c., s. g. 1022.	—	—	0.0			
					2 hour post-glucose.	+	+	+ 0.1	+		
					3 hour post glucose.	—	...	......	...	......	Very suspicious case.
E. S.	31	Trace, 1898. Trace, 1899.	None made.	Urine frequently negative for glucose. Developed tabes. 1904, after 100 gm. cane sugar 0.42 per cent. glucose.	24 hour spec., 885 c.c., s. g. 1024.	—	—	0.0			
					2 hour post-glucose.	+	+	+ 0.4			
					3 hour post-glucose.	+	+	+ 0.1	+	— 0.2 per cent.	Suspicious case.
H.	38	Trace, 1898. 0.56 per cent , 1900.	None made.	Healthy. June, 1904, 0.67 per cent., frequently traces found by physician, frequently absent.	24 hour spec.. 1450 c.c., s. g. 1026.	—	—	0.0	...	......	July, 1906, one hour after lunch sp cimen showed 0.8 per cent. glucose.
					Before glucose.	+	+	+ 0.3	+	— 0.5 per cent.	
					2 hour post-glucose.	+	+	+ 2.5	+	0.1 per cent.	
					3 hour post-glucose.	—	+	— 0.4	—	— 0.35 per cent.	Diabetes.
W.	36	Trace, 1899. Trace, 1900.	1899, normal.	Healthy. Sugar frequently found in traces since, frequently absent.	24 hour spec., 1010 c.c., s g. 1033.	+	+	+ 0.1	...	......	Family history, diabetes.
					Before glucose	+	+	+ 0.05	+	— 0.1 per cent., no ozanon.	
					2 hour post-glucose.	+	+	+ 2 7	+		
					3 hour post-glucose.	—	+	— 0 2	—	— 0.225 percent	Diabetes.
B.	31	Trace, 1900.	1890-98 normal 4 times, 1898.	Healthy. Sugar frequently found in traces, frequently absent.	9 hour spec., 935 c c., s. g. 1026.	+	+	+ 0.8			
					Before glucose.	+	+	0 0	+	— 0.12 per cent., no ozanon.	
					2 hour post-glucose.	+	+	+ 3 5	+		
					3 hour post-glucose.	+	+	......	+	......	Diabetes.
P.	48	0.33 per cent., 1901; 2 days later, 0.37 per cent.	1894.	Healthy. Sugar occasionally present or absent.	24 hour spec., 2000 c.c., s. g. 1028.	+	+	+ 0 3			
					Before glucose.	+	+	+ 0 1	+	— 0.15 per cent	
					2 hour post-glucose.	+	+	+ 1.6	+		
					3 hour post-glucose.	+	+	+ 1.6	+	— 0.18 per cent.	Diabetes.

With these particular ends in view we have undertaken this research, and we have been given an opportunity for the partial elucidation of the questions at issue in the extensive material very courteously placed at our disposal by the medical directors of one of New York's large insurance companies.

We selected from the company's records all of the New York City cases showing a glycosuria of 1 per cent. or less during the years 1900 and 1901, choosing these slight glycosurias so as to exclude, if possible, all diabetics. This gave us 69 cases, of which 24 could not be traced. Of the 45 left, 20 joined cheerfully in our research, while 25 refused to do so. However, we did ascertain that these diffident 25 were alive, and at least 13 of them, whom we interviewed personally, were apparently in good health.

The preceding table summarizes the histories of the 20 cases which form the basis of our work, and the results of our examinations and experiments carried out on them during the summer of 1906.

In regard to their previous history we see that 8 subjects had shown normal urine on examination either by an insurance company or their own physician at periods varying from three weeks to ten years before sugar was first discovered. Twelve subjects had never had their urine examined before. We have records of sugar having been found once in three men and twice in two men prior to 1900. Of the 15 remaining cases, with their first record of sugar in 1900 and 1901, 7 showed it again at some subsequent period; 17 of the 20 subjects showed normal urine (office specimens) at least once after the sugar was first discovered. Some of them, it is true, observed spasmodically a restricted diet. The remaining 3 gave normal twenty-four-hour specimens in 1906.

We may, therefore, group all of these cases together as cases in which a transient spontaneous glycosuria was first discovered some time between 1895 and 1901, and in no case could we then say that anything more than a transient spontaneous glycosuria existed. Of course, we do not know the exact date at which the glycosuria was first present, nor can we say positively that the initial glycosurias were not the result of a diet excessively rich in sugar, although as far as the memories of the subjects served, this particular supposition was negatived by our 20 cases.

There is no doubt that these men showed glucose in their urine in 1900 or 1901. A specimen of urine was passed in the presence of the insurance physician and was examined by him with Fehling's solution. If it reduced Fehling's it was sent to an experienced chemist, who re-tested it with Fehling's solution, with phenyl hydrazin, by fermentation, and with the polariscope.

We obtained twenty-four-hour specimens from 14 men, twelve-hour specimens from 5, and small, before and after lunch, specimens from 1. We found all but 2 of the subjects in excellent health, 1 suffering from chronic nephritis and 1 from tabes; 9 had gained in weight, 9 had remained stationary, and 2 had lost weight. No evidences of arteriosclerosis could be detected except in the case of nephritis, and aside from the urine there was nothing to suggest diabetes in any of them. In the group of 5 cases showing sugar previous to 1901, 2 had developed a constant slight spontaneous glycosuria (one of them having a family history of diabetes) and could be called diabetic, although the glycosuria was the only symptom present. Of the 15 remaining subjects who had first shown sugar during 1900 and 1901, 2 had developed a constant slight glycosuria, but showed no other diabetic symptoms.

Thus 4 of the 20 subjects developed mild diabetes in from five to eight years. The 16 remaining showed normal twenty-four-hour specimens. At a later period 18 of these men were each given 100 grams of glucose during the forenoon, about three hours after breakfast. We then secured from them specimens of urine passed two and three hours after the ingestion of glucose. No food was taken during this time. Our results are also included in the foregoing table.

The interpretation of these results introduces the subject of the relation between transient spontaneous glycosuria and glycosuria *e saccharo*.

THE RELATION BETWEEN TRANSIENT SPONTANEOUS GLYCOSURIA AND GLYCOSURIA E SACCHARO.

By this we mean the relation existing between the ordinary spontaneous glycosuria, as exemplified in the 20 cases just considered, and the glycosuria produced in cases of this kind by the ingestion of 100 grams of glucose or saccharose. We hope to show that we have in this estimation of the sugar tolerance a means of determining at a relatively early date which cases of spontaneous glycosuria will become diabetic.

Some technical terms and the details of experiments with alimentary glycosuria must now be considered. By the assimilation limit for any sugar, a term first used by Hofmeister,[9] we mean that quantity of sugar necessary to be ingested in order to produce a trace of sugar in the urine. Individual susceptibility is an important factor in fixing this assimilation limit, for the same quantity of sugar will not produce glycosuria in all healthy people, even if given proportionately to the body weight (Moritz).[10] There also are transitory conditions, entirely physiological, which lower this limit (H. Strauss).[11] The assimilation limit for glucose varies from 150 to 200 grams (von Noorden).[12]

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[9] Archiv f. experimentelle Pathologie and Pharmakologie, 1889, xxv, 249.

[10] Verhandl. X Congress f. innere Medizin, p. 125.

[11] Deut. med. Woch., loc. cit.

[12] Disorders of Metabolism and Nutrition: Diabetes. Eng. trans., 1905, p. 33.

Whether the sugar is given on an empty stomach or not has a bearing on the result. Glucose is carried from the intestine by the portal vein and deposited in the liver as glycogen. If the intestine is suddenly overloaded with glucose a very small portion may be absorbed by the thoracic duct (von Noorden)[13] and excreted by the kidneys, without going through the liver. Thus a slight glycosuria, occurring after glucose taken on an empty stomach, does not necessarily mean that there is a superabundance of glycogen in the liver.

If it takes 150 to 200 grams of glucose to produce glycosuria in a normal man, production of glycosuria by 100 grams would prove conclusively that the assimilation limit for glucose was lowered at that particular time. H. Strauss[14] carried out experiments on 50 subjects, giving each 100 grams of anhydrous glucose dissolved in 500 c.c. of water, on an empty stomach, and produced slight glycosuria in but 4. On repetition of the experiment, however, these 4 were negative.

The assimilation limit for cane sugar is the same as that for glucose, 150 to 200 grams (von Noorden).[15] Sometimes, owing to changes in the intestinal ferments, cane sugar, or saccharose, fails to split up and is excreted as cane sugar, the liver being unable to appropriate it in this form. However, if glucose is excreted after the ingestion of 100 grams of cane sugar, we can infer that there is an excess of glycogen in the liver; so, whether we conduct our experiments with cane sugar or glucose, the results are the same, provided we test always for glucose.

The results of experiments with cane sugar on healthy people are somewhat contradictory. Linossier and Roque[16] found glucose in the urine in 5 cases after the ingestion of from 50 to 100 grams of cane sugar on an empty stomach. Worm-Müller[17] found in 1 case glucose in the urine after the ingestion of 50 grams of cane sugar. Moritz[18] states that he always found glucose in the urine after large quantities of cane sugar had been taken.

There are few recorded experiments with cane sugar on normal people, using the standard dose of 100 grams, and not giving it on an empty stomach, so that it seemed advisable to carry out an experiment under proper conditions. Ten of the third-year students of the Cornell University Medical College kindly volunteered for the experiment. Each showed a normal twenty-four-hour specimen and a normal specimen just before taking the cane sugar. At 10 A.M. 100 grams of chemically pure cane sugar dissolved in 350 c.c. of water was administered to each of them.

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[13] Handbuch. der Pathologie des Stoffwechsels, von Noorden, p. 29.

[14] Deut. med. Woch., loc. cit.

[15] Disorders of Metabolism and Nutrition: Diabetes. Eng. trans., 1905, p. 34.

[16] Arch. de méd. exper., 1895, vii, p. 228.

[17] Pflüger's Archiv f. Physiologie, 1884, Band xxxiv, p. 576.

[18] Loc. cit.

Specimens were taken one, two, three, and four hours afterward, no food being taken in the interim. The following table shows the results:

October 30, 1906.		Fehling's solution.	Polariscope.	Ozanon.	Fermentation.	Rotation after fermentation.	Remarks.
			per cent.			per cent.	
W.	10.00 A.M.	—					
	11.00 "	—	— 0.3	—			
	12.00 M.	—	— 0.2	—			
	1.00 P. M.	—	— 0.1	—	—		
	2.00 "	—	— 0.1	—	—		
S.	10.00 A.M.	—	— 0.1				
	11.00 "	—	— 0.2	—	—		
	12.00 M.	—	— 0.225	—			
	1.00 P. M.	—	— 0 1	—			
	2 00 "	—	— 0.1	—			
S.	10.00 A.M.	—	— 0.3	—	—		
	11.00 "	—	— 0.3	—			
	12.00 M.	—	— 0.3	—	—		
	1.00 P. M.	—	— 0.3	—			
	2.00 "	—	— 0.1	—			
F.	10.00 A M.	—	0.0				
	11.15 " 12.10 P. M.	—	— 0.1	+	+	— 0.18	No invert sugar.
	1.00 "	—	— 0.1	—	—		
	2.00 "	—	— 0.1	—	—		
O. B.	10.00 A.M.	—	0.0				
	11.00 "	+					
	12.00 M.	+	— 0 5	+	+	— 0.4	
	1.00 P. M.	—	— 0.1	—	—	……	Gave Seliwanoff's reaction for levulose.
	2·00 "	—	— 0.1	—	—		
C.	10.00 A.M.	—	— 0.1				
	11.15 "	—	— 0.3	—	—		
	12.30 "	—	— 0 3	—	—		
	2.00 P. M.	—	— 0.2	—			
	3.00 "	—	— 0.2	—			
B.	10.00 A.M.	—	— 0 3				
	11.00 "	—	— 0.2	+	+	— 0.25	
	12.00 M.	—	— 0.2	+			
	1.00 P. M.	—	……	—			
	2.00 "	—	— 0.15	—			
K.	10.00 A M.	—	— 0.15				
	11.00 "	—	— 0.2	—			
	12.00 M.	—					
	1.00 P. M.	—	— 0.2	……	……	……	No invert sugar.
	2.00 "	—	— 0.1				
Z.	10.00 A.M.	—	— 0.05	—			
	11.10 "	—		—			
	12.10 P. M.	—	— 0.25	+	+	—0.3	
	1.15 "	—		+			
	2.00 "	—	0.225	—			
B.	10.00 A.M.	—	0.0				
	11.00 "	—	0.0	+	+		
	12 00 M.	—	— 0.15	—	—		
	1.00 P. M.	—	— 0.1	—	—		
	2.00 P. M.	—	— 0.15	—	—		

We found that 3 cases showed about 0.05 per cent. of glucose, and 2 gave traces of glucose. These quantities of glucose are so small that they are negligible. Nine showed an increase in the levorotatory substances, the percentage varying from 0.2 to 0.4. The Kowarsky modification of the phenyl-hydrazin test was used, and the precipitate examined at the end of one hour. The percentage of glucose was estimated by the Schmidt and Hansch polarizing saccharimeter.

We see, therefore, from H. Strauss' experiments on 50 normal people with glucose, and from our experiments on 10 normal people with cane sugar, that the ingestion of 100 grams of either substance will not produce more than mere traces of glucose in the urine of healthy individuals.

A large amount of work has been done with alimentary glycosuria, both *e saccharo* and *ex amylo*, its object being a more exact knowledge of the nature of diabetic glycosuria, but the results have been rather contradictory. The only reference von Noorden makes to these investigations, as far as we can find, is in his *Lectures on Diabetes*,[19] where he says: "They (the experiments with alimentary glycosuria) did not fulfil this expectation, but brought to light many points of interest." Naunyn[20] says that an alimentary glycosuria *e saccharo* may be a diabetic glycosuria, but not necessarily, and most frequently is not. If we find it present after 100 grams of glucose, a suspicion of diabetes exists. If the sugar exceeds 1 per cent., the suspicion is very urgent. Raphael,[21] as a result of his experiments, is constrained to think that these two forms (alimentary *e saccharo* and diabetic glycosuria) have no important difference. J. Strauss[22] says: "From all this I am obliged to conclude that the alimentary glycosuria *e saccharo* is also to be placed in the category of diabetic disturbances of metabolism." H. Strauss,[23] who worked chiefly with alcoholics and the traumatic and non-traumatic neuroses, concluded that in the majority of the conditions in which an alimentary glycosuria is noted a transient glycosuria is also frequently observed, and the glycosurias generally disappear at the same time.

One reason, perhaps, for these diverse opinions is that many of the experiments have been carried out in pathological conditions of the body, such as chronic alcoholism, paralysis, meningitis, etc., which have but a slight and limited relation to diabetes. The ordinary transient spontaneous glycosuria, however, presumably the earliest manifestation of diabetes and a most promising field for experimentation, has been comparatively neglected, probably owing to the scarcity of available material.

We have found only 7 cases of spontaneous glycosuria in the literature, in which the reaction to glucose or cane-sugar ingestion was ascertained. F. Raphael[24] reports 2 cases which show alimentary

[19] English trans., 1905, p. 38.

[20] Nothnagel's Specielle Pathologie und Therapie der Diabetes Mellitus, p. 19.

[21] Ztschr. f. klin. Med., xxxvii, p. 48.

[22] Ibid., xxxix, p. 292.

[23] Deut. med. Woch. Loc. cit.

[24] Ztschr. f. klin. Med. Loc. cit.

glycosuria ranging from 1 per cent. to 3 per cent., and in from one to two years developed diabetes. Von Noorden[25] reports 1 case showing 1 per cent. of alimentary glycosuria, which developed in one month a constant glycosuria. B. de Harn[26] also reports 1 case which gave an alimentary glycosuria of 0.9 per cent. and in less than a year developed diabetes. J. Strauss[27] reports 3 cases, previously showing an alimentary glycosuria, but gives no percentages nor the subsequent history of his cases.

All of the above cases of spontaneous glycosuria, excepting the 3 of J. Strauss, showed a marked alimentary glycosuria and developed diabetes in a short time. The experimental technique in these cases, however, varied widely, and, therefore, no basis is afforded for comparison with our results.

We shall now consider the results of our experiments with the production of alimentary glycosuria, in our 18 cases of transient spontaneous glycosuria. These were conducted, as explained in the previous section, under similar conditions, during the summer of of 1906. (See first table.)

Four showed an alimentary glycosuria of from 1 per cent. to 3 per cent., these being the 4 diabetics, additional evidence of the correctness of our diagnosis; 5 showed from 0.1 per cent. to 0.4 per cent. of glucose, and 2 showed only traces of glucose, not sufficient being present to estimate quantitatively; 7 were negative. Of the 5 showing measurable quantities of glucose, cases C. K., M. G., and E. S. we strongly suspect are diabetic at the present time, because of their past history and their decided reaction to the test. In view of our experiments with cane sugar on the 10 students, and H. Strauss' experiments with glucose on 50 normal people, we attach no importance to the results on the test of those 2 cases which reacted only to the phenylhydrazin test.

If we regard alimentary glycosuria *e saccharo* as a disturbance of metabolism essentially diabetic in nature, we may, in the light of this test, again summarize our results as to the subsequent history of 18 subjects of transient spontaneous glycosuria, and say that 4 have become diabetic, 3 are probably diabetic, and 2 are suspicious cases, while 9 are not diabetic; 1 of the 2 who refused the test is a chronic nephritic, and the other never showed sugar after 1901, although examined repeatedly, and gave a normal thirteen-hour specimen. So there is little doubt that these 2 also are free from diabetes. This makes 11 altogether free. These results, 4 diabetics and 5 probable diabetics out of 20 cases, contrast decidedly with our previous results stated at the end of the first section, when we found that but 4 had become diabetic and 16 were presumably healthy.

It will be remembered that 18 of our subjects were examined again some time after sugar had first been found, and 11 of them showed a glycosuria on this second examination. These 11 included the 4 diabetics, 4 of the suspicious cases, and 3 cases now normal. This exemplifies the prognostic value of a repeated detection of traces of sugar.

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[25] Verhandlungen der XIII Congress f. innere Medizin, 1895, p. 180.

[26] Archiv f. Verdauungskrankheiten. Loc cit.

[27] Ztschr. f. klin. Med., xxxix, p. 202.

We also found that 5 of these 20 cases, in addition to glucose, excreted a levorotatory substance varying from 0.2 per cent. to 0.4 per cent. (as estimated by the saccharimeter), which substance did not ferment, gave a positive reaction to Bial's test and the phloroglucin test, and with the spectroscope showed a broad band between C and D. It seems fair to assume that this was combined glycuronic acid. Three of the cases showed no glucose, but did show from 0.2 per cent. to 0.5 per cent. of this same substance. It will be noted that the sugar excretion reached its maximum in the two-hour post-glucose specimens.

The insurance company carried out experiments in alimentary glycosuria during 1903 and 1904 on all sugar cases, and it will perhaps be of some help if we compare their results in the spontaneous glycosurics with our results.

THE INSURANCE COMPANY'S EXPERIMENTS. Tests were made by the company over a period of eighteen months. All applicants having a history of sugar were given, in a small number of cases, 100 grams of glucose, in the greater number, however, 100 grams of cane sugar, dissolved in 300 c.c. of carbonated water, at some time in the forenoon or afternoon. Then a specimen of urine was obtained approximately three hours after, but this time varied considerably. No restrictions as to eating during the interim were made. As explained above, there is no difference in the results of the glucose or cane-sugar test, provided we consider it positive only in the presence of glucose in the urine.

Obviously, tests which were conducted under such varying conditions possess much less value than ours, and deductions therefrom are correspondingly limited. We selected from the records all cases showing a spontaneous glycosuria of less than 1 per cent. to which the test was applied within one year of the discovery of sugar. These were men who presented no symptoms of diabetes and believed themselves in excellent health at the time of the initial glycosuria. These formed a group of 19 cases of ordinary spontaneous glycosuria similar to our 18 cases, except that our cases did not receive the glucose test until at least five years had elapsed. The following table summarizes the company's results:

Name.	Date initial glycosuria.	Quantity of sugar.	Time elapsing between initial glycosuria and test.	Urine before glucose.	Urine after glucose.
		per cent.			
M. W. Z.	April 21, '04	0.48	4 months.	1024, no sugar.	1021 —0.48 per cent.
R. L.	April 4, '03	0.83	3 months.	1024 "	1004 —0.56 "
	April 7, '03	0.56			
S. B.	Jan. 3, '05	0.57	23 days.	1023 "	1022 —0.25 "
G. S. A.	June 14, '04	Trace.	8 months.	1026 "	1028 —0.5 "
T. B. B.	Nov. 25, '04	Trace.	3 months.	1016 "	1020 —0.44 "
A. A. K.	Jan. 17, '05	0.28	2 months.	1025 "	1030 —0.25 "
M. J.	June 20, '04	0.18	2 days	1020 "	1020, negative.
H. A.	Dec. 10, '04	0.22	1 day.	1025 "	1022 "
	Jan. 19, '05	0.33			
G. H.	Aug. 5, '04	Trace.	3 months.	1025 "	1025 —0.33 per cent.
	Aug. 6, '04	Trace.			
B. E.	Jan. 18, '05	0.31	12 days.	1012 "	1006, negative.
C. B. A.	Jan. 15, '05	0.25	16 days.	1023 "	1018 "
E. M.	Nov. 3, '04	0.25	24 days.	1022 "	1021 "
H. H.	June 25, '03	0.42	10 days.	1020 "	1020 "
	Nov. 21, '04	0.67			
E. M. G.	July 11, '04	0.33	1 day.	1018 "	1022 "
J. M.	Dec. 30, '04	0 33	2 days.	1008 "	1020 "
A. S.	Nov. 14, '05	0.35	11 days.	1020 "	1020 "
J. T. H.	Feb. 3, '05	0 44	2 months.	1016 "	1032 "
A. K.	Jan. 26, '05	0.24	14 days.	1020 "	1020 "
	Jan. 21, '05		2 days.		

Thus we see that 7 cases showed an alimentary glycosuria ranging from 0.2 per cent. to 0.6 per cent., and 12 were negative. It is interesting to note that these 7 positive cases received their glucose or saccharose test from three weeks to eight months after sugar was first discovered, while all of the 12 negative cases except 2 received their tests a few days after the discovery of sugar.

The only legitimate deduction to be made from these experiments is that at least 37 per cent. of a group of spontaneous glycosurics showed an alimentary glycosuria *e saccharo* within one year of the initial discovery of sugar.

As regards the time element in alimentary glycosuria we may here mention that F. Raphael[28] concluded, as a result of his experiments with the glucose test on susceptible patients (including his 2 cases of spontaneous glycosuria), that the sugar excretion varies in quite wide limits at different times, or may entirely disappear. These conclusions are supported also by the work of H. Strauss,[29] Arndt,[30] Rosenberg,[31] and Marin,[32] with the same tests in various diseased conditions.

Now, we found that 9 of our 18 cases showed a measurable alimentary glycosuria at the end of from five to eleven years, and the insurance company found that 7 of their 19 cases showed alimentary glycosuria at some time within one year of the first discovery of sugar. The obvious question is, Would the same cases showing alimentary glycosuria *e saccharo* within one year of the initial glycosuria also show it at the end of five years? In other words, do we possess in this test a means of deciding, at an early date, which cases of spontaneous glycosuria will show a continued disturbance in their sugar metabolism and ultimately develop diabetes?

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[28] Ztschr. f. klin. Med. Loc. cit.

[29] Deut. med. Woch. Loc. cit.

[30] Deut. Ztschr. f. Nervenheilk., 1897, x, p. 419.

[31] Inaug. Dissertation, 1897: Alimentary Glycosuria, etc.

[32] Berl. klin. Woch., 1897, Nr. 52.

Before answering this, let us sum up the data we have at hand:

1. Fifty per cent. of a group of spontaneous glycosurics showed alimentary glycosuria *e saccharo* at the end of five years, and included all of the diabetics and probable diabetics.

2. Of our 17 cases of spontaneous glycosuria reëxamined at some time between 1895 and 1906, 58 per cent. showed sugar again, 8 of these developing diabetes or probable diabetes.

3. In 2 of our cases, E. S. and W. E., we are able to trace the different phases of disturbed sugar metabolism from the first appearance of sugar to the development, years later, of possible diabetes. Thus spontaneous glycosuria in 1898 and 1901, again in 1899 and 1902, alimentary glycosuria *e saccharo* in 1903 and 1904, and again in 1906.

4. We know, from the insurance company's experiments, that at least 37 per cent. of a group of spontaneous glycosurics showed alimentary glycosuria *e saccharo* within one year of the initial discovery of sugar.

In other words, 50 per cent. of a group of spontaneous glycosurics showed a disturbed sugar metabolism (reaction to glucose) five years after the first discovery of sugar; 58 per cent. of the same group showed a disturbed sugar metabolism (reappearance of sugar in the urine) two or three years after sugar was first discovered. 37 per cent. of another group of spontaneous glycosurics showed a disturbed sugar metabolism (reaction to glucose) within one year of the first discovery of sugar. Finally, we can, in 2 cases, trace the disturbed sugar metabolism through the entire five years.

These various facts may be explained by an assumption which readily suggests itself, that is, the division, broadly, of spontaneous glycosuria into two types: (1) A class essentially diabetic from the outset, in which sugar recurs, which shows constantly glycosuria *e saccharo,* and which at the end of five or more years has become diabetic or probably diabetic. (2) A class, quite harmless, in which sugar does not recur after the first weeks and which does not show a glycosuria *e saccharo* except perhaps during the first few weeks, and does not develop diabetes. The glucose test, therefore, would seem to give important help in differentiating these two provisional types of spontaneous glycosuria at a relatively early date.

SUMMARY. 1. At the end of five years 20 per cent. of a group of spontaneous glycosurics had become diabetic, 15 per cent. had become suspicious cases, and 10 per cent. somewhat suspicious; 55 per cent. had remained free from diabetes.

2. Eight of eleven cases of spontaneous glycosuria, in which sugar recurred, became diabetic or probably diabetic.

3. The alimentary glycosuria arising from glucose or cane sugar, provided the test is properly conducted and repeated at intervals, affords a valuable aid to prognosis in cases of spontaneous glycosuria. A positive test is of much more value than a negative test.

4. Alimentary glycosuria *e saccharo* is essentially diabetic in its nature.

Von Noorden,[33] as a result of his belief that a transitory glycosuria "is in most cases the warning signal of later diabetic disease, the outbreak of which may not occur for many months or even years," advises in all of these cases "a moderate reduction of carbohydrate food and especially a complete exclusion of sweet comestibles." We found at the end of five years but 45 per cent., at the most, of our cases had become diabetic. If it is possible, as we believe, to detect these cases at a relatively early date, it would not seem necessary to maintain a permanently restricted diet in *all* cases.

If a small quantity of sugar is found in a patient's urine and there is no history of overindulgence in sweets, he should be placed on a restricted diet and his reaction to the glucose test ascertained frequently during the next six months. If the reactions are positive his restricted diet should be continued, even though the spontaneous glycosuria has not recurred. If the reactions are negative and sugar does not reappear, a restricted diet would seem superfluous.

The conflicting opinions as to the essential nature and diagnostic worth of alimentary glycosuria may be harmonized by more observations on the course of cases of spontaneous glycosuria, and by the frequent determination of their reaction to the glucose test.

We wish to express our thanks to Doctors T. W. Hastings and Mortimer Warren for several analyses.

INFANTILE SCURVY, ITS MANIFESTATIONS AND DIAGNOSIS.

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Infantile scurvy is a comparatively new disease, having been recognized for less than twenty years. The first edition of Osler's *Practice of Medicine*, which appeared in 1892, mentions the disease, but earlier text-books do not allude to it. In the periodicals devoted to pediatrics reports of cases of infantile scurvy began to appear about 1890. Even at the present time physicians do not always keep in mind that scurvy may occur in infancy, so that they often fail to recognize the disease. Because of the resemblance of some of its symptoms to those of other diseases it may be of interest to consider its various manifestations and to emphasize its diagnostic features. Undoubtedly scurvy has occurred in infants for centuries, but it has not been sufficiently frequent for extended or careful study until the last two decades. During this period the very common use, by those who can afford it, of dried proprietary infant foods, or of sterilized milk, has greatly increased the number of such cases.

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[33] Disorders of Metabolism and Nutrition: Diabetes. English trans., 1905, p. 127.

Formerly the condition was called acute rickets; but, strange to say, this form of rickets was almost always complicated by so-called "ulcerative stomatitis." Only last summer I heard of a typical case of infantile scurvy which was exhibited by a professor, at one of the great German schools of medicine, as a case of acute rickets with ulcerative stomatitis. So, even at the present day some of our German colleagues are barely aware of the existence of the disease in England and the United States: many of them claim that they do not have scurvy in Germany.

The French have become interested in looking for the disease, and for the last two or three years there have been frequent reports of isolated cases, occasionally of half a dozen cases; so they are finding it not a rare disease.

The English were the pioneers in the study of the disease and in the recognition of the distinctions between rickets and scurvy. Cheadle was of the opinion that "acute rickets," so called, was a compound of rickets and scurvy; Gee called the condition "osteal or periosteal cachexia;" while Barlow, after a study of eleven cases, with postmortem examinations of two, showed the essential nature of the disease and gave it the name "infantile scurvy." The Germans, with their tendency to fasten a proper name to every disease, call it Barlow's disease

Barlow summarized the chief features as follows: "(1) Predominance of lower-limb affection with (*a*) immobility, with the knee slightly flexed and thigh rotated outward, often pseudoparalysis; (*b*) excessive tenderness; (*c*) general swelling of the lower limbs, due for the most part to subperiosteal hemorrhage; (*d*) skin shiny and tense, but seldom pitting and without undue local heat; (*e*) thickening of the shaft of the bone, made out when the swelling has disappeared; and (*f*) liability to fracture near the epiphysis. (2) Swelling of the gums, varying from definite sponginess down to minute transient ecchymoses. The conditions mentioned constitute the chief diagnostic criteria between scurvy and rickets in children. To these should be added (3) the tendency to hemorrhage either into the skin, subcutaneous tissues, mucous membranes, or rarely into the viscera, and (4) the definite and rapid improvement upon antiscorbutic diet."

Inasmuch as concrete instances are the best means of illustrating the disease, the following cases have been selected from among those I have seen in private practice to show both the more common, as well as the unusual, phases of the malady. I have used only private and consultation cases for the reasons that their histories are more reliable and the patients were more carefully followed afterward than can be done in hospital and dispensary work.

CASE I.—*Pain, tenderness, and swelling in the lower extremities; blueness of the gums; pseudoparalysis of the legs. Developed while taking sterilized milk.*

Alice J., eight and one-half months old, was seen at Far Rockaway August 27, 1901, in consultation with Dr. Lewengood. The child had been fed from birth on modified milk made up and sterilized at the Walker-Gordon Laboratory. Barley-water had been used in the modification. Five weeks previous to the time I saw her, when just over seven months old, she began to have pain and tenderness in the legs, crying when the diaper was changed. Next she did not move the legs as usual, and later not at all; later still there was swelling of the lower part of the left thigh just above the knee. Three weeks previous to my visit a prominent orthopedic surgeon saw her and made the diagnosis of scurvy; he advised discontinuing the barley-water and substituting pasteurization of the milk for the sterilization; it was also advised that a splint be applied to the affected knee. For the week preceding my visit the baby had been getting worse, the milk meantime being both peptonized and pasteurized.

On physical examination I found the child ashy pale, with a dry, scaly skin; it did not cry, but lay motionless unless disturbed. There was intense tenderness in both lower extremities; so great was the fear of being touched that the child screamed out on the mere approach of her favorite nurse. A heavy tread near the bed would make her scream from the slight jarring. Both thighs were swollen, the left one being ten and one-half inches in circumference, the right nine and one-half inches. The bluish, tense, fusiform swelling of the left thigh was much like that of a rapidly growing sarcoma; but it was exquisitely tender, while the right thigh was less so. There was no crepitus to be felt in either thigh, that is, no sign of epiphyseal separation such as not infrequently occurs when the subperiosteal hemorrhage has existed for so long a time. The liver and spleen were markedly enlarged; there were no signs of rickets. There were no teeth, but the gums over the upper incisors were swollen and blue.

There was no question about the diagnosis, but only in regard to the treatment. Pasteurization of the milk, formula 3 per cent. fat, 6 per cent. sugar, and 1.5 per cent. proteids, was stopped, and orange-juice and beef-juice ordered to be given, a teaspoonful of each three times a day. In twenty-four hours the tenderness was less acute, and in a week the swelling was perceptibly smaller, the baby using her legs freely. Six weeks later there was still much enlargement of the left femur, a smooth exostosis remaining, comparable to the result of cephalhematoma in the newborn. It was fully twelve months before the difference in the size of the two femora disappeared entirely.

Five weeks ago I saw the child. She is now six years old, weighs forty-six and three-quarters pounds, has perfect use of her legs, and is a splendid specimen of health. There are absolutely no evidences of her serious illness except a persistent slight enlargement of the spleen.

In this case the diagnosis was correctly made by the attending physician and the orthopedic consultant. A splint was applied, but the dietetic fault was not entirely eliminated.

Case II.—*Tenderness of the thighs and ankles, swelling of the gums, and pseudoparalysis. Developed on pasteurized milk.*

James McC., eight and one-half months old, was first seen June 1, 1903. He had been a healthy child, fed from early infancy on modified milk from the Sheffield Farms laboratory. The laboratory directed the feeding, and changed the formula as necessary, I was told; moreover, I learned that the food had always been pasteurized. The formula being taken was: 4 per cent. fat, 5.6 per cent. sugar, 2.5 per cent. proteids, pasteurized.

For two weeks the baby had cried when the diaper was changed. The main complaint was that the baby was very fretful and did not use its legs naturally; the mother feared that the baby had paralysis. There was no digestive disturbance except constipation; no blood in the urine or in the stools.

Physical examination revealed a pale, flabby child covered with a purplish rash and clammy perspiration; there were no petechiæ. The gums about the four upper and two lower incisors were swollen and blue. The baby lay uncomplaining unless touched, but cried whenever the arms and legs were moved. He did not use the lower extremities at all, but moved the arms. There was great tenderness and some swelling of both thighs, the left thigh measuring seven inches, the right thigh measuring six. There was some tenderness and swelling above the ankles, and slight but distinct tenderness above the wrists. The heart was normal; there were a few coarse rales in the lungs; temperature 100.2° F., by rectum.

The pasteurization was discontinued, and the formula was 3.5 per cent. fat, 6 per cent. sugar, and 1.5 per cent. proteids ordered; also orange-juice, one-half ounce three times a day.

The next day it was reported that the baby was somewhat improved, being less fretful. The left thigh was one-quarter inch smaller and was less tender. On June 3 the left thigh was still smaller and the child brighter. On June 7 the child was playing with the left toe in his mouth. On June 8, one week after treatment was begun, the right thigh measured five and one-eighth inches, the left thigh six inches, a reduction of one inch in circumference during the week. Purée of potatoes was then added to the dietary.

Incidentally it was discovered that the baby was a masturbator by thigh rubbing. The mother averred that he would cry when caught doing this, and that he tried to conceal the act. Accordingly, a crutch was made to fasten to the thighs and keep them apart. Four weeks later the baby had gained three pounds.

It is to be noted that in this case the milk had simply been pasteurized, heated to 157° F. for twenty minutes, and no starchy food had been used.

Case III.—*Tenderness; blood-blebs over the gums; pseudoparalysis. Developed while taking milk that was not heated when made, but overheated when warmed for the bottle feeding.*

Alice McS., twelve months old, was referred to me by Dr. M. C. O'Brien, on July 11, 1903. The child had been nursed for two months, then weaned because the breast milk failed; was given milk and barley-water for a while, then milk made up with peptogenic powder; then Mellin's Food; and then modified milk from the Walker-Gordon laboratory, formula 3.5 per cent. fat, 6 per cent. sugar, 1 per cent. proteid, made up with one ounce of dextrinized barley-jelly; next, the formula was changed to 4 per cent. fat, 6.5 per cent. sugar, 1.5 per cent. proteid with barley-water. The laboratory milk was neither sterilized nor pasteurized; but careful questioning elicited the information that the milk for several weeks had been heated in a saucepan to warm it before feeding. In this way it was probably heated to at least the temperature of pasteurizing, as a rule, and to a higher temperature often. The baby's digestion had improved, but there had been latterly some vomiting, so that the Walker-Gordon milk was stopped and a home-made formula used, as follows: 11 ounces of Borden's mixed milk, the whites of two eggs, 8 teaspoonfuls of Robinson's barley, boiled in sufficient water to make 40 ounces altogether.

The chief complaint was that the baby cried when the stockings were being put on or when the legs were moved; the mother thought that the baby had "paralysis partly." Moreover, the baby had a stool about every time it took food.

The child was very pale, an ashy pallor, and was covered with perspiration; it was merely fretful if let alone, but cried out when either the legs or arms were touched; there seemed to be tenderness of the spine and the baby would not sit up. The upper and lower gums bulged and were blue. There was little or no swelling to be found. There was a bruise-like discoloration on the dorsum of the right foot. Routine physical examination revealed signs of bronchitis in the chest.

The food was continued exactly as above save for the heating, which was discontinued; orange-juice and beef-juice were added. In one week the baby had improved so that it could be moved without crying; there was less tenderness of the legs and arms, so that the child began to move the legs and tried to sit up. The vomiting ceased and there were six to eight small normal stools per day.

Following this the baby had bronchopneumonia and quotidian malaria. She recovered entirely from the scurvy symptoms in two weeks. I saw her in April, 1906, and operated on her for adenoids. She is now a fine rosy specimen of health, and shows not a trace of the disease.

CASE IV.—*Bloody urine; tenderness of the extremities; bleeding from the bowels. Developed on pasteurized peptonized milk.*

Dorothea O., ten months old, was referred to me by Dr. L. Emmett Holt on July 24, 1900. The baby was not nursed at first, but was fed on sterilized modified milk, one-third milk and two-thirds water; then barley-water, then peptonized milk. She did not thrive, so that at two months she was given to a wet-nurse for three weeks. Becoming worse the nursing was discontinued and peptogenic milk pasteurized was again tried—this time with fair success, as the baby gained so that the weight at seven months was 17½ pounds. At seven and one-half months blood was detected in the urine, which was being passed in very small quantity. There was no pain in the joints nor any change in the gums. The diet was changed and for three weeks the baby was given oatmeal gruel; during this time the urine became normal. When eight and one-half months old the peptogenic milk was made up with oatmeal gruel and pasteurized. At once blood and albumin reappeared in the urine, and this persisted. A simple modified milk was then tried and the stools became normal. Next the yellow, smooth, well-digested stools began to contain bright, sometimes frothy blood: the blood was both around and mixed in the stool. It had been noticed that, for three weeks before coming under my observation, handling the baby made it cry.

When examined July 24, 1900, the temperature was 101° F.; the respiration 34, the pulse 132, the weight 14 pounds. The baby was fairly nourished, but of ashy-gray color, due to an intense anemia with purplish mottling all over the body, giving a livid appearance; the skin was covered with clammy perspiration. The child's condition was very poor, bordering on collapse. The fontanelle was even with the general surface of the scalp, and one inch by one inch. There was one bluish spot one-quarter by one-half inch behind the right ear. Two lower central incisors had pierced the gum; around them the gums were dark purple and swollen. There were numerous fine, purple petechiæ on the chest, abdomen, and back. The child cried after moving the lower extremities; the arms were moved without pain. There was no swelling of the joints nor of the legs, but there was great tenderness just above both ankles, particularly the left, but no tenderness above the knees. The urine contained albumin and red blood cells, but no casts.

A diagnosis of scurvy was made, and so serious was the child's condition that it was feared she might die before the parents reached home.

The treatment instituted was to put the child on raw modified milk suitable for a three months' baby; to give also 1 ounce of orange-juice and one-half ounce of beef-juice, these to be doubled on the second day.

For two days the baby cried almost incessantly; on the third day the crying ceased and the legs could be moved without pain; and the blood disappeared from the urine and from the stools. On the sixth day the profuse sweating ceased and the gums returned to normal condition. Eight days after treatment was begun the baby had gained one pound in weight.

The first sign of scurvy in this case was the bloody urine; following this came tenderness of the legs and blood in the well-digested stools; next were seen the blood blebs around the teeth, and finally the petechiæ. The case emphasizes the marked tendency to hemorrhage from the mucous surfaces and into the skin which is produced by this nutritional vice.

Case V.—*Blood in the stools and swollen; bluish gums. Developed on a weak milk mixture made up with starchy food.*

Ogden N., aged twelve and one-half months, was seen August 30, 1903. The patient had been under my care from the age of eight months. There was a marked rheumatic family history and a tendency to eczema; the child also had moderate adenoids not requiring removal. When nine months old the child contracted pertussis from his brother, and the whole family at once went to the country about May 15. At about the end of the whooping cough he developed a severe diarrhœa, on account of which the Walker-Gordon milk was stopped for a few days and broth given instead; then the milk was resumed in small quantity given with barley-water and alternated with broth. Every time the milk was increased to over one-fourth of the total food the stools became loose and filled with curds and mucus. On this account the milk was kept low, and for five weeks the child took 10 ounces of top milk (Walker-Gordon) raw, with 32 ounces of barley-water. A proprietary milk food (Nestlé's) was added to make up the deficiency in proteids. With care the milk was increased to 22 ounces of mixed milk, and the child did well for two weeks, when suddenly the stools became loose, at first only two movements in twenty-four hours, but both containing a little blood. On the following morning, August 31, the baby had six movements, all containing some flecks of blood. That afternoon I saw the child in the country and learned that there had been some loss of appetite and fretfulness for several days before the bowel trouble began. Naturally, I thought the case was merely a summer diarrhœa—an enterocolitis with a little blood. The temperature was only 100° F. by the rectum, and when the stools were shown they did not give evidence of intestinal indigestion, no curds, no mucus, no foul odor; moreover, the blood was not mixed with the stool nor with any mucus. The baby was very fretful and further examination revealed nothing until I finally inspected the mouth. The gums were then seen to be much swollen and blue all around the four upper incisor teeth. There was no swelling nor tenderness at the extremities.

I stopped the barley-water and all starchy food at once, and ordered 2 ounces of orange-juice and 2 ounces of beef-juice; the food formula was made 2 ounces of mixed milk, raw, 2 ounces of lime-water, and 6 ounces of water. The next day the gums were better, being much less swollen; in two days the stools were free from blood and only two in twenty-four hours. The unboiled milk was rapidly increased and the child became speedily well.

In this case if there had not been any bulging and blueness of the gums I should have considered it ordinary enterocolitis. The rapid disappearance of the blebs of blood around the upper incisors when antiscorbutic measures were used, together with the subsidence of the diarrhœa and the disappearance of blood from the stools when the orange-juice and beef-juice—both well-known laxatives—were given, showed the case to be one of scurvy with hemorrhage from the intestinal mucous membrane.

Case VI.—*The spinal joints remaining tender after the usual symptoms disappeared. Developed while taking milk and oatmeal gruel dextrinized by Cereo, a preparation of diastase.*

Van Wyck R., aged fourteen months, was first seen July 19, 1901. The family history was markedly rheumatic. The infant, healthy at birth, had been nursed entirely for two weeks; nursing was then supplemented by bottle food for a time and weaning was complete at six weeks. The successive foods taken by the baby were: (1) Mellin's Food with milk and water, the milk not being boiled; (2) malted milk, made up with boiled water; and (3) raw milk with barley-water dextrinized by Cereo. These various changes were made up at the end of the second month, when milk with oatmeal gruel and Cereo was begun. By the ninth month the proportions reached were about equal parts of oatmeal gruel and milk. After two and one-half months on this diet, when eleven and one-half months old, the baby stopped using his legs, and seemed to have pain when the legs were lifted to change the diaper. He kept his knees stiff. Next blue spots and some swelling appeared below the knees; but none over the knee-joint. There was also blue swelling of the gums and a bruise-like discoloration of the soles of the feet. When the child was at its worst, the gums would bleed. After about two weeks in this condition, because of almost complete loss of appetite, the mother changed the diet by adding beef-juice, soft-boiled egg, and mutton broth; orange-juice also was given occasionally. The main symptoms remaining when I first saw the child were inability, or rather refusal, to sit up and an ashy pallor.

On examining the baby, I found many evidences of rickets in the enlarged epiphyses at the wrist and the lateral furrows at the lower part of the chest; there was also marked enlargement of the spleen and liver. The child used its legs quite well and the tenderness of the legs had disappeared. There remained, however, the discoloration of the skin on the shins and soles, due to subcutaneous hemorrhage. The most important symptom was crying when raised up to a sitting posture; the baby could not be persuaded to sit up. Careful examination revealed marked tenderness of all the spinal joints. There was no knuckle and only slight spasm of the spinal muscles upon lateral flexion. The gums showed blueness, softening, and bulging between the upper incisor teeth. The diagnosis was plainly scurvy and rickets.

A proper *régime* was established, and fresh food with raw milk and animal fats were given; the carbohydrate food was discontinued. Within a week the mother reported the greatest improvement in the baby's temper, comfort, and animal spirits. The child was not seen again for two months, inasmuch as he lived up the State. When seen the "bruises" had entirely disappeared; the child sat alone, would creep, stand, and push a chair about the room. All tenderness had disappeared from the joints of the spine.

The spinal tenderness in this case was quite marked. I had never read of nor seen such a condition in scurvy; but it seemed comparable to "typhoid spine." Dr. Henry Ling Taylor tells me he has seen this spinal tenderness in cases of scurvy, and that he reported such a case.[1]

CASE VII.—*Stiffness and swelling of one knee simulating arthritis. Developed while taking a proprietary food with heated milk.*

James S., Jr., aged nine months, was seen November 18, 1904. This child was also one of my own patients, so that the feeding history is accurately known. He had been perfectly healthy and digesting his food well until the hot spell in July, 1904. At that time, when five months old, he was taken acutely ill while in the country with vomiting and diarrhœa. The trained nurse, who had been with the child since birth, managed the case very well, and in a few days the child was taken to the seashore. At first the food was simply whey; then alternated with Nestlé's Food. From August 1 to October 15 the baby had an increasing quantity of fresh milk until he was taking equal parts of milk and water, with the addition of Nestlé's Food. The baby gained in weight from 14 pounds 11 ounces, the lowest weight, to 18½ pounds on September 30, 3½ pounds during the two months of August and September. On October 14, just two and one-half months from the time the milk and Nestlé's Food were begun, I received word from the nurse that the baby seemed to have colic at night; was very fretful, awaking about every hour. The family was in Tuxedo for the autumn, so I tried to advise by telephone. The baby's appetite was poor, and he had lost

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[1] Archives of Pediatrics, September, 1894.

5 ounces during the week. Orange-juice and dilution of the food were ordered. On October 17, three days later, the report came that the baby was still having colic, though the stools seemed all right. The next day I saw the baby in Tuxedo, and found that the nurse had already observed the fact that the child did not care to lie down, and that he did not use his legs as much as usual. On examining the stools I found them normal; the abdomen revealed nothing; the heart and lungs were negative; there was no swelling or bleeding of the gums nor signs of teeth coming. The urine was normal. The temperature was 99.4° F. On examining the lower extremities, I found some swelling and tenderness of the right knee. Measurements showed the right knee 8½ inches, left knee 8 inches. The baby had lost 11 more ounces in two days, then weighing 17 pounds.

The probable diagnosis was clear, but the causative element seemed lacking. On careful questioning of the nurse I learned that the hot prepared proprietary food had always been added to the fresh milk without having been cooled. The previous food was stopped and formula of 3½ ounces of fresh mixed milk, 1 ounce of lime-water, 1 tablespoonful of milk sugar, and 3½ ounces of water was substituted. The orange-juice was increased to 4 ounces daily.

The next day the so-called colic was less and the baby began to move the leg; the swelling immediately began to subside, and was gone in three days more. In one week the baby had gained 3 ounces. On November 16, when the family returned to town, the left knee measured 7½ inches and the right knee 7¾ inches; there was absolutely no tenderness.

The prompt subsidence of the tenderness and swelling when the heating of the milk and the proprietary food were discontinued and the fruit-juice increased showed the case to be due to a dietetic error and the condition to be scurvy.

SYMPTOMATOLOGY. It will now be interesting to consider the results of the most important investigation of the disease yet made. This is the collective investigation of infantile scurvy by the American Pediatric Society. Next to Barlow's writings, this report is the most valuable contribution to this subject.

Statistics of 379 cases were collected, the reports coming from 138 physicians all over the United States and Canada. Summarizing the results, this investigation showed that the disease appears with equal frequency in male and female infants, for the most part between the age of seven and fourteen months. Nearly two-thirds of the cases appear during the latter half of the first year, and about two-fifths during the eighth and ninth and tenth months. The youngest patient was three weeks old, a breast-fed baby; the oldest patient was nine years of age. The cases reported were mostly from private practice with good hygienic surroundings.

One might almost say that the hygiene of the food, so far as sterilizing and great care against germs are concerned, is altogether too good. We rarely observe scurvy in tenement patients or those that are recklessly fed; since the taste of this bit of fruit or of potato or other table food prevents the development of scurvy that might occur from the use of patent foods or even boiled or sterilized milk, which is quite commonly used by the tenement-house population.

As regards causation, the investigation showed that the food seemed to be the most important etiological factor. Strange to say, in 10 cases the symptoms came on when breast milk had been the sole food from birth. The breast milk examined chemically from some of these cases proved to be totally inadequate for the needs of nutrition, and the infants were victims of mal-nutrition in these cases before the signs of scurvy appeared. But by far the greatest number of cases developed while the infants were being fed on some proprietary or starchy food, with or without milk, and on sterilized, pasteurized, or peptonized milk. The lack of freshness, or of fresh proteid particularly, seemed to be the main factor in each case in which the proprietary and starchy foods or the heated milk were used; in the breast-fed infants there seemed to be a deficiency of these vital proteids.

As to the manifestations of the disease, the symptoms first seen were, in order of frequency, pain and tenderness of the extremities, sponginess or puffiness of the gums, disability, anemia, cutaneous hemorrhages, swellings, and rarely hematemesis, hemorrhage from the rectum, and hematuria. Proptosis, protrusion of the eyeball, due to hemorrhage into the back of the orbit, and often hemorrhage under the conjunctiva, have been noted as the first symptom by English observers.

Dr. Richard Kalish, in a personal communication, speaks of 3 such cases he had seen; in 1 of these a corneal ulcer developed. Dr. Snow, of Buffalo, reported 1 such case at the 1905 meeting of the American Pediatric Society.

The pain and tenderness were usually noticed only on motion or handling; at times they may be so exquisite that the child screams when anyone approaches the bed or even looks at the child, for fear of being touched or jarred. The tenderness and swelling are found most frequently in the lower extremities, especially over the lower part of the femur or the lower part of the tibia. Very rarely the upper extremities alone, one or both, may be affected; generally the arms are involved after the thighs or legs. Fracture, really separation of the epiphysis, occurred only a few times.

The gums escaped in only 16 out of 379 cases. There were all degrees of involvement, and it must be borne in mind that the gums are frequently affected even when there are no teeth yet cut through. There were cutaneous hemorrhages in over 50 per cent. of the cases; bleeding from the mouth or gums in about 25 per cent., from the nose in 9 per cent., from the bowels in 10 per cent., and hematuria in about 6 per cent. of the cases.

Symptoms of rickets were present in varying degrees in nearly one-half of the cases.

PATHOLOGY. The constant findings at autopsy have been subperiosteal hemorrhage in the diaphysis of the long bones, especially near the epiphyseal junction. There may be separation of the epiphysis and fracture. The hemorrhage may extend into the adjacent muscles and rarely into the knee-joint. There may be hemorrhages into the subcutaneous tissues, the mucous membranes, the skin, and more rarely into the viscera, particularly the spleen.

COMPLICATIONS. There is always a concomitant anemia and a general cachexia which increases with the duration of the disease and the internal or external bleedings. Enterocolitis and gastroenterocolitis are not infrequent complications. Fortunately the proper treatment of the scurvy usually has a marked beneficial effect on the intestinal condition. Most to be dreaded are the pulmonary complications, lobar pneumonia, and bronchopneumonia. Most of the fatal cases had pneumonia or succumbed to the progressive anemia and cachexia.

DIAGNOSIS. For diagnosis it is only necessary to keep in mind that scurvy does occur in infants, especially from seven to fourteen months old, and that its main features are pain, tenderness, and disability of the extremities, with swelling of the gums and a tendency to hemorrhage. If additionally there is the history of the child having been fed for a long time upon a sophisticated food or sterilized milk the diagnosis is certain.

DIFFERENTIAL DIAGNOSIS. The list of diseases for which scurvy has actually been mistaken is a long one; it is interesting that error in diagnosis has usually been made by a physician or surgeon working in some special field and seeing, consequently, few cases of infant feeding. The cases I have cited from my own records show early features which might have related to almost every type of specialist. Certain symptoms of scurvy in infants have been repeatedly mistaken for those of the following diseases:

1. *Rheumatism.* The fact that rheumatism is rare under two years of age, and almost unknown under one year, should be presumptive evidence against rheumatism in young infants. Moreover, in scurvy there is usually no fever or local heat in the swollen part, and the swelling is along the shaft of the bone primarily instead of in the joint.

2. *Rickets, especially Acute Rickets.* The intense pain, the swollen, bleeding gums, and the tendency to hemorrhage will indicate scurvy to be present even when there is coexistent rickets. The best of treatment is also of great aid in the differential diagnosis. In cases of scurvy and rickets the scorbutic symptoms disappear at once, while the rachitic symptoms persist for some months at least.

3. *Arthritis.* Gonococcic arthritis must be considered in the very young. Septic or pyemic arthritis must be considered if there is tenderness near several joints, and especially if there is profuse sweating. The correct diagnosis will be made upon careful physical examination, which shows the swellings to be in the shafts of the bone near the joint rather than in the joint or periarthritic structures. The condition of the gums will clinch the diagnosis.

4. *Paralysis, especially Anterior Poliomyelitis.* The drop-toe and loss of power are often suggestive; but the gradual development of the disability, the tenderness, the enlargement near the epiphysis, and the fact that the foot and leg can be moved, though reluctantly, show that the loss of motion is only the pseudoparalysis of scurvy.

5. *Syphilitic Epiphysitis.* On account of the epiphyseal swelling, tenderness, and pseudoparalysis, also on account of the frequent enlargement of the liver and spleen in scurvy, syphilitic epiphysitis is closely simulated. The preference of syphilis, however, is for the upper extremities, while scurvy usually affects the lower; moreover, the characteristic eruption of syphilis is absent, while the bruise-like marks, petechiæ, and swollen gums of scurvy are present.

6. *Sarcoma of the Femur.* Unfortunately there are many instances in which general surgeons have operated for sarcoma, when the swelling was unilateral, only to find a subperiosteal hemorrhage. The tenderness of the other limbs together, with the tumefaction of the gums, will correct the diagnosis.

7. *Osteomyelitis or Deep Abscess.* Goodhart and Still assert that the disability and swelling of the femur or the tibia has at times led surgeons to explore the bone for deep abscess. The longer history and the absence of fever, together with the swelling of the gums, should prevent this error.

8. *Purpura Simplex and Hæmorrhagica.* Purpura is to be distinguished by the absence of swelling of the long bones and of tenderness in the shafts of the bones

9. *Nephritis.* Since albumin with blood in the urine may be the first, and for a time the only, sign of scurvy the diagnosis of acute hemorrhagic nephritis has been made. The feeding history, together with the absence of fever, should make one suspicious. The tenderness, disability, and swelling of the extremities, together with the changes in the gums, confirm the diagnosis of scurvy. From nephritic œdema of the legs the differentiation is to be made by the absence of pitting on pressure, the disability of the legs, the great tenderness, and later by the changes in the gums.

10. *Dysentery; Ileocolitis, with Bloody Stools.* The diagnosis may be difficult, as in the case I have cited. The absence of fever, and of mucus or feces mixed with the blood, and the otherwise normal stools, will merely make one cautious about the diagnosis, until changes in the legs and gums occur.

11. *Intussusception.* At a discussion of this paper before the Hospital Graduates' Club of New York, Dr. J. F. Erdmann reported that he had been called one night by two physicians to operate for intussusception upon a child just under eight months of age. The child was apparently in a condition of shock, and was having cramp pains and bloody mucous stools. An exploratory operation seemed advisable, but during its course the infant went into collapse. While efforts at artificial respiration were being made, the gums were observed to be spongy and bleeding. The baby died. Further examination by daylight and close questioning into the previous feeding history proved without doubt that the child's bloody defecations were of scorbutic origin, no intussusception having been found upon operation.

In all the above cases, if the diagnosis is uncertain, the effect of treatment, the therapeutic test, is of the greatest value, inasmuch as the results show in forty-eight hours, and no harm can be done by waiting that long, as a rule. In a word, if in doubt, when the baby cries when the diapers are changed or the stockings are removed, stop the sophisticated food and give raw milk and orange-juice.

Prognosis. The duration of scurvy is variable. If the faulty diet is continued without modification the child fails and dies of exhaustion or from some complication. When proper treatment is instituted improvement is almost miraculous. No results in medicine are more brilliant and none so easy to obtain. Within a comparatively few hours a pitiable, suffering little paralytic is transformed to a contented baby, waving its arms and legs in the sheer joy of living.

Treatment. Only a word needs to be said. The cause is to be removed. We are to stop the use of devitalized food, and to give fresh milk, fruit-juice (orange, grape, or pineapple), beef-juice, raw egg albumen, and purée of potato, according to the child's digestive capacity. In infants over a year old, purée of potato and of green vegetables is especially satisfactory. To discontinue the sophisticated food is often all that is necessary; but cure is more rapid when the fruit-juices are added.

Scurvy in adults is now almost a curiosity of medicine; it is dying out because understood. Its causes and treatment are recognized by the laws of all maritime nations, so that nowadays the disease is seldom seen even in the great seaports of the world.

A similar history of infantile scurvy is likely to obtain, and only sporadic cases appear almost as curiosities. This will not occur, however, until that distant day when infants are no longer fed on patent foods, but on fresh cows' milk that has been produced under ideal hygienic conditions. We shall still have cases of infantile scurvy until physicians, as well as the laity, realize that heating or boiling milk is not a physiological but a therapeutic measure. Recently Dr. Arthur Jacobus has informed me that much of the milk sold in New York City by two of the milk companies has been already pasteurized or sterilized before delivery; this is done in order to evade the law against preservatives and the law against acidity or sour milk. Perhaps this fact may account for some of the cases of scurvy we see. To sterilize milk is often necessary, but it should be adopted rather as a mode of treatment than as a method of feeding; and it should not be continued indefinitely, any more than should arsenic, on the ground that it is a special form of food for the nervous system.

THE TREATMENT OF FRACTURE AND DISLOCATION OF THE VERTEBRÆ.

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Fracture of the spine, alone or accompanied by dislocation, while not an every-day occurrence, is still sufficiently frequent in the experience of men actively interested in traumatic surgery to merit a discussion of its treatment. The question of operative interference is also an open one and the results of such interference seem to vary to a considerable degree. There seems to be altogether too much tendency on the part of surgeons to look upon the injury, when severe, as necessarily fatal, and to decline to operate on the ground that they are "surgeons, not executioners," a sentiment that is utterly detestable to the chances of progression along this line of work.

During the course of the past summer two cases came under my care which presented some extremely interesting and instructive points, and which led me to inquire into (1) the degrees of injury to the spinal cord which we may assume to exist with different degrees of injury to its bony envelopment, and (2) the cases as they appear clinically.

The Pathological Condition. Presuming a fracture to exist the following seems to be a reasonable classification of the resultant conditions:

Pathological Class I. Fractured fragments slightly or not at all depressed; membranes and cord not injured.

Pathological Class II. Fractured fragments depressed; membranes torn, with hemorrhage about the cord, but no injury to the cord itself.

Pathological Class III. Fractured fragments considerably depressed, or the fracture accompanied by dislocation; membranes lacerated, hemorrhage about the cord, contusion of the cord itself, and slight hemorrhage into its substance.

Pathological Class IV. Fractured fragments much depressed and usually accompanied by dislocation; lacerated membranes, hemorrhage about the cord, severe laceration of the cord itself, with hemorrhage into its substance.

Pathological Class V. A combination of the above conditions in which the dislocation is the more prominent feature and in which the cord is caught and compressed by the dislocated fragments, with or without contusion or partial laceration.

These divisions are made arbitrarily, differ principally in the degree of injury, and any of the conditions may exist as the result of direct or of indirect violence.

The factors in the foregoing conditions which are productive of symptoms and the symptoms they produce are these:

1. *Laceration of the cord,* causing an immediate loss of the power of movement, or sensation, or both.

2. *Compression of the cord,* causing a gradually increasing loss of the functions of movement or sensation, or both; this compression itself may be either (*a*) immediate, due to a bony deformity established at the time of injury, or (*b*) delayed, due to the action of an unchecked hemorrhage started at the time of injury. If the former, there is very little interval between the time the injury is received and the beginning of the onset of cord symptoms; if the latter, this interval may be long or short, but is always present.

3. *Laceration and compression of the cord,* producing the immediate symptoms of laceration combined with the more tardy symptoms of compression.

Hence we may assume that a case of severe back injury, in which the patient presents the symptoms of cramps in the legs, burning and tingling of the skin of the legs, followed by an increasing loss of the functions of movement and sensation in those parts, is a case of compression of the cord. Again, if a man with a fractured or dislocated spinal column has immediate loss of motion and sensation in his legs, we may safely say that such a symptom-complex points accurately to laceration of the cord. Finally a patient who presents the symptoms of immediate motor paralysis of the legs, followed by a gradual loss of sensation in the paralyzed parts, is clearly one in which there has been laceration of the motor area and compression of the part presiding over the function of sensation.

Thus the cases as they appear clinically divide themselves for our consideration into three classes:

Clinical Class I. Pure compression cases, that is, those which present the history of injury to the vertebral column, and locally the signs of fracture or dislocation, followed at an interval by an increasing paralysis.

Clinical Class II. Pure laceration cases, that is, those which present the unmistakable signs of fracture or dislocation, accompanied by immediate loss of sensation and power of movement of any or all structures whose site of spinal-cord control is located at or below the point of injury.

Clinical Class III. Combination cases, that is, those which, together with the gross signs of structural injury, show an immediate loss of sensation or power of movement, followed by a progressive loss of the functions not immediately destroyed.

TREATMENT. A careful consideration of the mechanics of the conditions likely to be present, and the mechanical means we have for their correction, will point the way to proper treatment.

Compression cases may owe their symptoms to one of several gross mechanical conditions:

1. Immediate bony compression may be due to (*a*) dislocation or (*b*) depressed fractured laminæ.

If due to dislocation we have the remedy in our hands: an open operation with suitable manipulation will reduce the dislocation and relieve the compression. If due to fractured and depressed laminæ, the offending fragments may be raised or removed entirely, at the discretion of the surgeon.

2. Delayed compression may be due to hemorrhage (*a*) about the cord or (*b*) into the cord.

In the former case the bleeding would naturally come from the vessels of the membranes and would create compression by its limitation by depressed laminæ: the remedy suggests itself, and a proper laminectomy together with control of the hemorrhage will afford the relief desired. In the latter case the hemorrhage into the cord would be, in all probability, merely one of the several factors entering into the general aspect of those cases which I have designated as combination cases.

Laceration cases, pure and simple, exist, from the standpoint of treatment, only when the cord is torn completely across, and all sensation and power of movement of structures physiologically below the particular segment under suspicion, are immediately abolished. Such cases are nearly always rapidly fatal and require the exhibition of such purely humanitarian methods of treatment as are afforded by the proper use of morphine. A condition of total cord destruction, at any level, justifies non-interference. Any injury to the cord of a less extent does not, in my opinion, carry with it any such justification whatsoever.

The combination cases are the most interesting, inasmuch as they present great opportunity for the saving of life, and, in addition, are those most frequently met with. Here we have a deformity produced which is of such a character that part of the cord is immediately lacerated and the remainder compressed. The duty of the surgeon is plain. We are powerless to repair the destroyed area. Suture of the cord is beautiful in conception but, unfortunately, absolutely worthless in practice. Compression can be relieved, and an open operation in which the surgeon attacks mechanical difficulties with mechanical means will remove the factor of compression and allow the uninjured portion of the cord segment to regain its functional activity.

Thus far I have purposely avoided discussion of the special advisability of operation in cases in which the injury affects certain different regions of the cord. I believe that any case into which the factor of compression enters, or may be expected to enter, should be operated upon, and I do not believe that the special segment of the cord involved should influence the surgeon either for or against operation. An injury to the cord even at the level of the lowest dorsal vertebra is surely sufficiently grave to require surgical intervention, and the higher the location of the injury the greater the demand for the relief which operation alone affords. The location of the injury affects prognosis seriously, but not indications for treatment. Finally, as regards the time of operation, delays are dangerous, perhaps fatal. The necessity for accurate diagnosis does not exist. By all means operate at once.

The following is a brief record of the first case I have to report:

John M., aged forty-three years, a plumber by occupation, while at his work August 13, 1906, fell twenty feet from a scaffolding, and struck upon his head. He was rendered unconscious. An ambulance surgeon applied suitable dressings to his several injuries and transferred him to Fordham Hospital.

He was admitted at 6 P.M., put to bed, and examined by a member of the interne staff. There were several large lacerated scalp wounds, under one of which, in the occipital region, was a linear fracture of the vault of the skull, which was not depressed, but which apparently ran to the base. In addition he had a simple fracture of the left patella. He was conscious and rational, generally contused, in shock, and complained of great pain in the neck and lower limbs. He was catheterized and three ounces of clear urine obtained. His numerous wounds were dressed, splints were applied to his injured leg, and then, unfortunately, his spinal injury was overlooked; he was given morphine and went to sleep. Upon admission his temperature was 98°, pulse 88 and small, and respirations 26.

He was next seen about 9 A.M., August 14, fifteen hours after his accident, and was found to have entire motor and sensory paralysis extending downward from a horizontal line two and one-half inches below the clavicles; complete anesthesia of the skin of both arms except over an area corresponding to the deltoid muscles; paralysis of the extensor muscles of both arms and forearms; a tendency to tonic contractions of the flexor muscles of the arms; and a fracture of the base of the skull was evidenced by extensive double subconjunctival ecchymosis. His respirations were entirely diaphragmatic and he had paralysis of the bladder and the rectum, evidenced by urinary continence and anal relaxation. All reflexes over the area of anesthesia were abolished.

I first saw the patient at 2 P.M. August 14, and found him in the condition indicated above. At that time his temperature was 102°, pulse rate 100, and respirations 30. His face had the peculiar waxy, expressionless appearance common to cases of spinal-cord injury, and he was exceedingly weak. He had pain in his contracted biceps muscles and in his neck; otherwise he was without sensation of any sort. It looked as though the man had but an hour or two to live; temperature increasing, respirations becoming more rapid; he was weakening rapidly. It was already twenty hours since he had received his injury. He had, in addition, a serious head injury, and altogether the case appeared absolutely devoid of hope. I felt it was my duty to let him die in peace. He received stimulation of various sorts, and in the morning of August 15 his condition was markedly better. It was decided to give him what chance he had.

At 10 A.M., forty hours after his injury was received, he was put on the operating table in a right lateroprone position, and chloroform administered. I made an incision five inches in length with its centre over the fifth cervical vertebra and carried it down to the spinous processes. The muscles were freed and retracted laterally, giving a good exposure of the spinal column. There was fracture of the spinous process of the fourth cervical vertebra, and a dislocation forward and downward of its body upon the body of the fifth cervical. The fractured spinous process was removed, together with a portion of its corresponding lamina, with rongeur and chisel. There was a small rent in the membranes and no subarachnoid hemorrhage. By the removal of the portion of the lamina I had established a point upon which I could exert leverage.

One assistant was delegated to make traction upward upon the head, another to press backward upon the throat, and a third to press downward directly upon the spinous process of the fifth cervical vertebra. When all were ready, I inserted a curved periosteal elevator into the spinal canal, between the membranes and the posterior portion of the canal formed by the dorsal parts of the fourth and third cervical vertebræ, and at a signal we all "heaved together," and the dislocation was reduced without any trauma to the cord itself. All bleeding was checked, the muscles were drawn together slightly in the median line by interrupted catgut sutures, the fascia united with a running suture of chromic gut No. 2, and the skin closed with silkworm-gut and silk. A small rubber-tissue drain was introduced to the site of fracture. A cast was immediately applied to his head and shoulders, and the patient was placed upon a water-bed. The time required for operation was forty-five minutes, and the patient left the table in better general condition than when he was put upon it.

He recovered consciousness in about twenty minutes and immediately began to move his arms. He had regained sensation over the entire dorsal surface of his arms and forearms and also over a small area corresponding to the middle third of his biceps muscles. There was a noticeable improvement in the depth and character of the respirations, and the tonic spasms of the flexor muscles of his arms were no longer present.

In two hours his temperature had reacted to 100° and his pulse was 100 and of fairly good character. From this point on he gradually became weaker, and died, eight hours after operation, of respiratory paralysis, his heart continuing to beat for several minutes after respiration had apparently entirely ceased. No autopsy was had.

This case seems to me to be one of pure compression. He certainly had no laceration of the cord, else we would have had some immediate evidence of it at the time of his admission; that there was no hemorrhage about the cord I demonstrated at the time of operation. The deformity was very marked and was easily sufficient to have caught the cord and gradually squeezed out its life. I cannot help feel that, had this patient been carefully watched and the first symptom of compression noted, operation at that time would undoubtedly have saved the man's life, and in all probability have restored him to complete functional activity.

My second case has had a more fortunate termination, and this in spite of the fact that his injury was more severe. The history is as follows:

Joseph D., about thirty-five years old, a carpenter by trade, was leaning out of a window of a building then in the course of construction, when he was struck on the back by an iron beam which weighed about three hundred pounds and which fell ten feet. He dropped back into the room unconscious. He regained his senses almost immediately and found himself unable to move his lower limbs. An ambulance carried him to Fordham Hospital. His temperature on admission, August 28, 1906, at 9 A.M., was 99°, his pulse 96, his respirations 32.

He was a well-nourished man. His face bore the peculiar waxy, expressionless look common to cases of this nature; the heart sounds were feeble and the pulse was small; respirations were shallow. The neck and upper extremities were normal. He was conscious and rational. Being unable to void urine he was immediately catheterized and several ounces of slightly pink urine was obtained. An enema, given to him shortly after admission, was returned slowly and without feces. He was unable to move his lower limbs; had a girdle sensation at the umbilicus, and irregular spots of anesthesia and paresthesia over the lower portion of his abdomen, and both thighs, legs, and feet. The knee-jerks were entirely absent. Priapism was present. He complained of tingling and burning in the lower part of his abdomen and in the thighs, legs, and feet, and intense pain in his back. On a line with the tenth rib there was a depression in the median line of the back, and a point of great tenderness.

I saw the patient at 10.30 A.M., two hours after the accident. He was in about the condition described above except that the anesthesia was becoming more complete. I advised operation. At 1 P.M. the patient was very weak, temperature 97° by rectum, pulse 72 and very small, and respirations 26. He was completely anesthetic from the umbilicus down. At 2.30 P.M. he consented to operation. Immediately before he was put upon the table he was again catheterized and four ounces of bloody urine was obtained.

At 3 P.M., six hours after the time of the injury, he was taken to the operating-room, placed upon the table in a right lateroprone position, and chloroform was administered. I made an incision six inches in length, in the median line of the back, with its centre over the depressed area. Cutting down to the spinous process the muscles and fascia were found ecchymotic. At the situation of the ninth dorsal vertebra there was a marked depression in the line of the spinous processes and those belonging to the eighth, ninth, and tenth dorsal vertebræ were evidently broken. The last two ribs on the left side were broken and the end of the twelfth rib was jammed into the region of the kidney on that side; the tenth dorsal was dislocated forward upon the ninth. Attempt at reduction at this point was defeated by impaction of the fragments. I removed the spinous processes of the tenth and ninth vertebræ, together with their corresponding laminæ, and found the membranes torn to pieces. From the position of the spinal cord it was evident that it had been pressed upon by the fractured laminæ just removed, and it appeared to be contused. My second attempt at reduction by manipulation and traction by means of strong bullet forceps, applied directly to the vertebral body of the tenth dorsal, was successful.

With the bodies of the vertebræ in line, the spinal cord in its proper place, and compression due to secondary dislocation guarded against by the removal of the laminæ of the tenth and ninth dorsal vertebræ, I considered that I had done all that could be done at that point. The region of the wounded kidney had ceased bleeding. The wound of operation was rendered as dry as possible, and the right and left erector spinæ muscles were drawn together in the median line with interrupted sutures of catgut. The fascia was sutured with chromic gut No. 2, and the skin closed with sutures of silkworm-gut and silk. A small rubber-tissue drain was inserted at the middle of the incision and carried down to the point of fracture. The patient left the operating-room in good condition and was immediately put upon a water-bed. The time required for operation was sixty-five minutes.

He rested nicely that evening and night, and the following day

I applied a plaster cast to his body, extending from the axilla to the knees, and removed the rubber-tissue drain. The experience of putting on the cast was very painful and trying for him, and during the time required for its application his pulse gradually quickened until it reached 180 to the minute. He also complained of great precordial pain. Hence his condition twenty-four hours after operation was not encouraging. His temperature was 102°, pulse 180, respirations 36. The urine contained much blood; twenty ounces in twenty-four hours by catheter. He was being vigorously stimulated, chief reliance being placed on saline and whiskey enemas, and strychnine hypodermically.

On the third day his condition was unchanged.

On the fourth day he was improving; by the eighth day his temperature was 98°, his pulse 92, and his respirations 20. Twenty-six ounces of pale, pinkish urine was obtained by catheter in twenty-four hours.

From this time there was no change until the tenth day, when he began to have irregular sensations of pressure and tingling in his legs and thighs.

On the eleventh day his general condition was good. He began to have painful sensations in his legs.

Later notes are as follows:

Fifteenth day. Wound dressed and found absolutely clean, with no stitch infection. Sensations better and improvement in left leg more rapid than in right. Urine now clear and normal in amount. Bowels moving after enemas. Catheter still necessary.

Ninteenth day. Patient beginning to have a little irregular temperature, since seventeenth day. Has slight cystitis. Bladder irrigations of boric acid twice daily.

Twentieth to twenty-eighth day. Cystitis has not grown any worse, but temperature has gradually increased in severity during the past week. Wound examined today and several spots of budding granulations along its course discovered. Apparently catgut infection. Openings slightly enlarged.

September 29, 1906, twenty-ninth day. Temperature normal. The cystitis is somewhat troublesome and tends to grow worse.

September 30. Patient anesthetized and an external urethrotomy done. Continual drainage afforded.

October 2. Patient in good condition. Cast removed.

Since that time the patient has steadily improved. Sensation is good in his entire left thigh, leg, and foot. His right thigh has some irregular spots where the sensation is not good. With the exception of these he has entirely regained his normal power of feeling. Motion has never shown any tendency to return. His skin is well nourished and there is remarkably little muscular atrophy. His wound of operation is entirely healed. His perineal wound is clean and granulating. He is not able to void urine. He feels well and looks well.

November 6. Patient was allowed to sit up in bed, and did so without experiencing any discomfort. In spite of the fact that he is paralyzed from his waist down, as far as movement goes he is particularly pleased with the fact that he is alive.

In regard to the operation itself there are some points which suggest themselves:

1. Nothing is gained and time is lost by a small incision.
2. No more bone should be removed than is absolutely necessary to provide proper exposure of the cord and a reduction of the dislocation.
3. If the membranes are found intact an aspirating needle will demonstrate the presence of blood beneath, and the membranes should not be incised needlessly.
4. The membranes should be closed, if possible, with a fine catgut or silk suture.
5. Drainage should be provided for any oozing that may take place from the wound of operation. A rubber-tissue drain is sufficient for this purpose and should be removed at the end of twenty-four hours.
6. An immediate external urethrotomy should be done and continual drainage provided for the bladder.

THE MOST FREQUENT HERNIA IN CHILDHOOD AND ITS SIGNIFICANCE.

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In 1904 I expressed my belief that the majority of herniæ in children are acquired and not congenital. Since then I have continued my study of the subject, but unfortunately have been obliged to delay until now the publication of the data bearing on the subject I have accumulated.[1]

If one turns to the work of those who regard these herniæ as being of congenital origin, and therefore often requiring active surgical interference, it is obvious that they take as the standard of their authority the fact that they have performed so many tens or hundreds of operations for the condition. Indeed, it is usually stated so in the early paragraphs of their papers. This is a very narrow point of view, which might almost be compared to an attempt to examine a landscape through a microscope instead of a telescope.

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[1] Lancet, August 20, 1904; later communications: International Clinics, 1905, ii, 15th series, p. 154, and Medical Review, 1905, viii, p. 569.

The problems of the origination of herniæ in children cannot be settled only by observations made at the operating table. Careful notes and records in the out-patient room should come first, and the results obtained in this way should be examined and checked in the operating theatre. It will then be found that there is a close relationship between the conditions of the parts noted in the theatre and certain facts in the clinical history observed in the out-patient room. Thus it becomes possible to understand and interpret the character of the cases.

With the aid of my clinical assistants, especially Mr. Howell Evans and Dr. Irving Pinches, a survey was made of the herniæ in the out-patients at the Great Ormond Street Hospital, who attended on my days. To these two gentlemen I owe many thanks for the work they have done. When we had completed about seven hundred observations it was felt that we had omitted to include a most important factor and that the whole work should be repeated The results of this collective investigation of the first series are summed up in the accompanying table:

I. FIRST SERIES OF 700 OBSERVATIONS.

	Per cent.
Right inguinal hernia	38.3
Right and left inguinal herniæ	10.8
Right and left inguinal and umbilical herniæ	8.0
Right inguinal and umbilical herniæ	12.0
Left inguinal herniæ	10.6
Left inguinal and umbilical herniæ	2.7
Umbilical herniæ	16.2
Epigastric herniæ	0.9
Femoral, right and left	0.15
Complicated by a ventral hernia between the divaricated recti	14.3
Single herniæ	66.0
Multiple	34.0

The above includes all the ordinarily recognized herniæ, but no account is taken of the median ventral hernia between the upper parts of the recti abdominis muscles, except that it was noticed casually that 14.3 per cent. were accompanied by this ventral hernia But as more and more observations were made, becoming convinced of the importance of this missing factor, we terminated the investigation after seven hundred observations had been made, and commenced a new inquiry on a wider plan and executed by a larger number of helpers, whose observations were "untainted" with my particular opinions on the subject. All concerned in this second investigation except myself were ignorant of the figures and fallacies of the first investigation, so that their observations were unbiased.

One very remarkable fact was shown by the first investigation; namely, that multiple herniæ are far more frequent in children than they are in adults. Single herniæ were found in 66 per cent.;

multiple herniæ in 34 per cent. This is suggestive either of the multiplicity of congenital sacs in one subject or of one factor which was the common cause for the herniæ present. But it was obvious that this survey was very incomplete, because we had omitted to take into consideration, as almost all previous authors have, the hernial protrusions between the divaricated recti.[2]

Like herniæ in other parts of the body, the ventral hernia between the divaricated recti is not necessarily obvious unless the abdominal muscles are thrown into action, such as is done when obtaining the impulse on coughing in adults. To do this in children requires some little skill and tact, so that the observations are not often made. The most generally applicable method is to place the child on its back on a table or its mother's lap; often it will begin to cry at once—when the ventral hernia will become obvious. If this is not so, its abdominal muscles may be stimulated by a finger or the handle of a pen. Should this fail, as it often does, the child's head should be supported with one hand, which at first raises it and then allows it to fall sharply backward two or three inches. Whilst this is being done the observer should watch the abdomen for the momentary contraction of the muscles; a carinate projection appears above the umbilicus if a median ventral hernia is present. In difficult cases other means may be required to demonstrate the protrusion. If care is taken, I believe that this hernia will be found more frequently than is indicated by my collective investigation of 2600 cases, in which it was noted in 24.21 per cent. In fact, one knows that the more carefully the observations are taken the more frequently will a median ventral hernia be found. For instance, two of those who helped me found the median ventral hernia present in 63 per cent. and 21 per cent. respectively, showing that the results vary with the observer, possibly depending on both the care and the time spent in making them.

Sir Frederick Treves[3] has stated that the sac, its coverings, and its contents are the three essential parts of a hernia. Recognizing that a hernial sac does not always contain viscus, a hernia may be recognized as being formed of a peritoneal protrusion which has separated some of the structures which form the abdominal walls; the formation of the peritoneal sac may or may not be accomplished by some abdominal viscus or viscera.

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[2] As my object was to obtain, in the second and larger survey, results independent of my private views I asked many to help. To them I must acknowledge my thanks: Dr. A. F Stabb, Obstetric Physician to St. George's and Queen Charlotte's Hospitals; Mr. G. E. Waugh, one of my surgical colleagues at Great Ormond Street; Mr. H. A. T. Fairbank, another of my surgical colleagues at Great Ormond Street and attached to Charing Cross Hospital; Mr. Howell Evans, of Great Ormond Street and Children's Invalid Aid Association, and Dr. Irving Pinches, my clinical assistants; Mr. Duncan Fitzwillian, Casualty Officer to Great Ormond Street; Dr. S. R. Gibbs, Mr. H. T. Gray, and R. T. H. Cox, of St. Thomas' Hospital; Dr. Vowe Johnson, of the Lambeth Schools Infirmary, and particularly Dr. Cursham Corner, for large and invaluable series of observations on the newly born.

[3] Text-book of Surgery.

Naturally these protrusions occur at the weaker regions of the abdominal walls: the inguinal, femoral, and umbilical regions. When the recti muscles are divaricated, the linea alba is stretched and thinned, allowing the peritoneum and perhaps the omentum and intestine to bulge forward. This bulging constitutes a true hernia having all the essential parts; and, as it is found almost exclusively in children, it is a peculiar character of their herniæ. Because it was neglected, my first series of observations which extended over two years and more was invalidated, and the second series was undertaken to remedy the defect.

As with all other herniæ, the median ventral hernia between the divaricated recti may be congenital or acquired. Differing from inguinal and femoral herniæ in the sac having no neck and communicating widely with the abdomen, its presence is demonstrable with its first appearance, which need not be so in cases of the others mentioned. The observations of Dr. Stabb and Dr. Cursham Corner have shown that it is a very uncommon, but not unknown, condition to find in the newly born; the former found it only once in 122 observations. Hence, there is little evidence in favor of its congenital origin. How then can it be acquired? As any given child can only digest and absorb a certain amount of nutriment, which varies with its every mood, any residue, whether an excess of good food or the remains of an indigestible meal, will "go bad" and ferment in the intestine unless expelled. For details of its fermentation I must refer the reader to another paper.[4] In this place it need only be stated that, amongst other products, gases are formed which distend the intestines, raise the intra-abdominal pressure, and, if not quickly got rid of, will dilate the belly, separating the component parts of its walls. To particularize the subject of communication, the ventral herniæ between the upper parts of the divaricated recti appear for two reasons: first, as the direct result of the increased intra-abdominal pressure; and second, as the indirect result of the continuance of the raised pressure upon the growth of the tissues of the linea alba.

The incidence of this ventral hernia at the different ages of life is of great interest. I am indebted to Dr. Cursham Corner for his observations in the early stages of life. He remarked that, on the average, the umbilical cord separated on the fifth day; when there was invariably some bulging of the umbilical cicatrix which lasted until the eighth day, when the scar became firm. The size of the bulging was dependent upon the length of cord left proximal to the ligature. He made 201 observations on children between birth and one year of age and found a median ventral hernia between the divaricated recti in 18.5 per cent., being more frequent amongst the older infants. At Queen Charlotte's Hospital, Dr. A. F. Stabb found only 1 case[5] of bulging between divaricated recti amongst 122 infants varying from one to thirty-nine days old. Amongst other observers, at the Great Ormond Street Hospital, bulging between divaricated recti was found in 34 per cent. of infants under six months of age and in 58 per cent. between six months and one year of age. These figures may be arranged as follows:

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[4] Some Surgical Results of Improper Feeding. Clinical Journal, June 27. 1906, p. 171.

	Per cent.
Queen Charlotte's Hospital (1 to 39 days)	0.8
East End Mothers' Home (1 to 365 days)	18.5
Great Ormond Street Hospital (under 6 months)	34.0
Great Ormond Street Hospital (6 to 12 months)	58.0

II. INCIDENCE OF A MEDIAN VENTRAL HERNIA BETWEEN THE DIVARICATED RECTI AT DIFFERENT AGES.

Years	Ventral herniæ.
1	30.0 per cent.
2	62.7 "
3	50.0 "
4	47.8 "
5	43.5 "
6	40.2 "
7	30.9 "
8	28.8 "
9	17.7 "
10	20.0 "
11	17.3 "
12	11.8 "
13 and 14	8.7 "

This table shows that the frequency of the median ventral hernia increases steadily from birth to attain its maximum in the second year of life; then it falls steadily until adult life is reached, when it is rarely seen. Therefore, it would appear that a median ventral hernia between the divaricated recti is an acquired character which becomes spontaneously cured as growth takes place. Its significance in connection with the formation of inguinal hernia is enormous. If one sees a house on fire and finds one story alight, it is natural to conclude that some incident has occurred which originated the fire. But if one finds two, three, or more stories alight, one concludes that they arose from one common cause and not that there were two, three, or more simultaneous fires kindled. Hence, if we find multiple herniæ, as in Table I, at least we may suspect that they have a common causation, though there is no proof that they are not all of congenital origin. But when in addition they are combined with a median ventral hernia between the divaricated recti, as in Table III, an acquired character known to be due to the raised intra-abdominal pressure, the probability is greater that they have a common and a similar origin. Thus, this form of ventral hernia becomes an indication of extreme etiological importance. In Table III is represented the percentage in which the presence of this ventral hernia was found at the different ages.

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[5] An infant one day old.

III. SECOND SERIES OF 2600 OBSERVATIONS.

	Of all cases.
Herniæ present in	32.7 per cent.

	Of all herniæ.
Right inguinal hernia	4.2 per cent.
Right inguinal hernia and median ventral hernia between the divaricated recti	4.7 "
Right inguinal and umbilical herniæ	0.8 "
Right inguinal and umbilical herniæ and median ventral hernia through the divaricated recti	2.3 "
Left inguinal herniæ	1.3 "
Left inguinal herniæ and median ventral hernia through the divaricated recti	2.1 "
Left inguinal and umbilical herniæ	0.4 "
Left inguinal and umbilical herniæ and median ventral through the divaricated recti	0.5 "
Umbilical herniæ	4.2 "
Umbilical herniæ and median ventral hernia through the divaricated recti	13.3 "
Median ventral hernia between the divaricated recti	61.3 "
Right and left inguinal hernia	1.0 "
Right and left inguinal herniæ and median ventral hernia through the divaricated recti	1.4 "
Right and left inguinal and umbilical herniæ	0.1 "
Right and left inguinal and umbilical herniæ and median ventral hernia through the divaricated recti	1.5 "
Epigastric herniæ	0.9 "

The median ventral hernia was found by itself in 61.3 per cent. of the cases of hernia; and when in combination as well, in 87.1 per cent.—a very strong indication of the probable origin of a very large number of the concomitant inguinal herniæ, and, as the congenital causation of them in certain cases is not disputed, of the composite, not simple, causation of herniæ.

This is strikingly brought out by the following figures which represent the percentage proportions amongst the various herniæ found. In Table V the herniæ accompanied by the ventral hernia between the divaricated recti is just twice as large as when unaccompanied—a fact which would suggest that pressure herniæ, that is, acquired, are twice as common as herniæ of congenital origin.

IV. FROM THE SECOND SERIES.

	Per cent.
Of all the 2600 cases of herniæ were found in	32.7
Median ventral hernia through the divaricated recti (with and without other herniæ)	24.21
Herniæ other than those between the divaricated recti	15.4
Herniæ unaccompanied by one between the divaricated recti	4.62

V. OF THE HERNIÆ THEMSELVES.

	Per cent.
Hernia accompanied by a median ventral hernia through the divaricated recti	25.8
Hernia unaccompanied by a median ventral hernia through the divaricated recti	12.9
A median ventral hernia through divarication of the recti; present alone	61.3
A median ventral hernia through divarication of the recti; alone and with other herniæ	87.1

Every variety of hernia is more common when associated with another between the divaricated recti than when alone or associated with any other variety of hernia.

There are two main theories of hernia formation: one, ably and vigorously advocated by many surgeons, amongst whom may be mentioned Mr. Stiles, of Edinburgh; Mr. Carmichæl, of Edinburgh; Mr. Murray, of Manchester; Mr. Clogg, of London, and especially Mr. Hamilton Russell, of Melbourne, who has named it the saccular theory, infers that herniæ appear on account of the sac being formed during development; the second, I believe first suggested by Mr. Arbuthnot Lane, in 1897, and amplified by me in 1904, in which it is argued that the majority of herniæ in children arise from the increased intra-abdominal pressure which results from the gas production of intestinal fermentation. On the latter theory, the hernia can be produced in either of two ways, or by a combination of both: first, the intra-abdominal pressure may protrude the hernia by direct pressure; second, and more commonly, the pressure acts indirectly by modifying the growth of the tissues, particularly in the weaker regions, facilitating the development of a hernia by direct pressure later. By the indirect action on its growth, the raised intra-abdominal pressure destroys the protective valvular action of the inguinal region, after which a hernia is formed. In very young children the valvular action may be rendered incompetent by the mere stretching of the soft growing tissues. Thus, the younger the child the shorter the time required for the development of a hernia. But the mere presence of a hernia in a child shortly after birth does not necessarily indicate its congenital origin; for instance, it is known even with the tougher tissues of adults that a hernia may appear in a few hours or days.

To recapitulate, a median ventral hernia between the upper parts of the divaricated recti is manifold the most common and characteristic hernia of childhood. Its importance has not been recognized owing to the fact that surgeons have made more observations in the operating theatre than in the out-patient room, and have, with few exceptions, never combined these two sets of observations. This median ventral hernia is almost invariably an acquired character and is directly related to the pressure of gas production by intestinal fermentation. Any hernia or set of herniæ in any situation is more frequent when accompanied by it than when alone (Tables III and V). Hence clinical experience shows that raised intra-abdominal pressure is directly related to the frequency of inguinal herniæ, and, it is suggested, probably also to their initiation. It has been further suggested that the true proportion of acquired to congenital herniæ is about 2 to 1 (Table V).

In the face of the fact that it is nearly impossible to recognize and distinguish the funicular variety of the congenital hernial sac from the acquired sac at an operation and even perhaps at a necropsy, there can be no certain knowledge of the formation of herniæ in children. Therefore, I would suggest that by means of the recognition of the presence and significance of the median ventral hernia, its frequency and associations, it is possible to introduce a strong probability into the question, which would seem to indicate that acquired herniæ are more common in children than those due to the presence of a congenital sac. This is not a mere academic question, as the line of treatment to be adopted must depend largely upon its appreciation.

THE CLINICAL RESEMBLANCE OF CEREBROSPINAL SYPHILIS TO DISSEMINATED SCLEROSIS.[1]

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The diagnosis between cerebrospinal syphilis and multiple sclerosis, while not usually regarded as difficult, may in some cases be very perplexing. Both multiple sclerosis and syphilis vary in their symptomatology according to the location of the lesions, so that difficulties in clinical diagnosis might be expected. In this paper no special reference is made to those cases of disseminated syphilitic lesions which resemble multiple sclerosis pathologically as well as clinically, nor is any possible causal relationship between the two diseases discussed.

A case in which a positive diagnosis between cerebrospinal syphilis and multiple sclerosis for a long time seemed impossible was under the care of one of us (Dr. Spiller) during several of his terms of service in the Philadelphia General Hospital, and was frequently presented in lectures to the students as probably one of multiple sclerosis. The patient died two weeks after the termination of one of these terms of service, and we are, therefore, indebted to Dr. James Hendrie Lloyd for the pathological material.

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[1] Read at a meeting of the Philadelphia Neurological Society, January 22, 1907.

P. B., aged twenty-eight years, was admitted to the Philadelphia General Hospital November 10, 1902, complaining of tremor, difficult locomotion, and a steady, constant pain in the legs just above the knees.

His father was living and well; his mother was dead of an unknown cause. Three brothers and one sister were living and well. The patient was married and the father of one healthy child. He admitted having had gonorrhœa when fifteen years of age and chancroid when about twenty years of age, but denied any history of syphilitic infection; subsequently, a few months before his death, he admitted having had syphilis. He denied excessive use of alcohol, and was always apparently strong and healthy. About six months previous to the onset of his present trouble he had his hands and feet frost-bitten while shovelling snow, but they were not much swollen, and he worked the next day as usual.

His present trouble began about two months before his admission to the hospital in 1902, when he noticed that when walking he would stagger and have pains in his legs. The pains were slight and not sharp or shooting in character. Three weeks after the legs were affected he noticed a beginning affection of the hands and arms, the nature of which is not recorded, unassociated with any pain, however. He stopped work about one week previous to his admission to the hospital, on account of repeated falls from his sweeping machine.

On his admission to the hospital it was noted that his gait was distinctly ataxic and that his station was very poor, so that he fell when he attempted to take the position of "attention" with his eyes open or closed. His irides reacted normally to light and in accommodation. He had general tremors of the limbs. His speech was affected and his tongue was tremulous. The knee-jerks were absent. Ankle clonus was slightly manifested and plantar irritation showed a slight extension of the great toe on the left side. The examination of the chest and abdomen was negative.

October 21, 1903. An ocular examination was made by Dr. E. A. Shumway; the ocular movements and the pupillary reactions were normal. Vision, O. D. $\frac{5}{20}$, O. S. $\frac{5}{15}$. The ophthalmoscope showed a pallor of the temporal half of the right optic nerve and a decided simple atrophy of the left optic nerve. The visual fields of both eyes were concentrically contracted, but especially that of the left.

November 12th. It was noted that the patient was exceedingly nervous and irritable. He did not have nystagmus. The upper extremities showed a coarse tremor on movement and he was unable to button his clothing. He had slight loss of voluntary power, the dynamometer registering 50 in each hand. With his eyes shut, he recognized correctly familiar objects placed in his hands. The tendon reflexes were apparently lost in the upper extremities, but the tremor made the examination difficult. His gait was ataxic, and he could not stand unsupported with his eyes open or closed. The knee-jerks were lost. Ankle clonus was present on each side, but the Babinski sign was apparently present only on the left side. He had no loss of sensation.

February, 1905. It was noted that the knee-jerks and ankle clonus were absent on both sides; that there was a marked intention tremor of the hands and a staccato speech.

February 20. An ocular examination was made by Dr. Charles A. Oliver. The right pupil was larger than the left. Both irides were sluggish to light, but prompt in accommodation and convergence. The movements of the external ocular muscles were good in all directions. The papulomacular wedge and the tissues in the optic nerve, downward and outward, were partially atrophic. The left optic nerve was more diffusely atrophied than its fellow. Efforts to outline the visual fields were unavailing owing to the patient's mental condition.

March 10, 1906. Notes made by Dr. Wm. G. Spiller. His condition was much as it had been for several years, only the man was much weaker than he had formerly been. He had difficulty in showing his teeth, and when he attempted to do so there were irregular movements about his mouth of the character of an intention tremor. He wrinkled his forehead well on each side, but was unable to draw up either corner of his mouth separately. When he closed his eyes there was a tremor of the muscles above the eyelids. This intention tremor of the muscles of the face was very striking and was a new sign. The movements of the eyeballs were free and there was no nystagmus. Sensation for touch and pain was normal in the face. The tongue jerked violently backward and forward when he attempted to protrude it, but when at rest it presented no tremor. The tremor of the tongue was an intention tremor. The masseter muscles contracted well. Speech was very difficult, tremulous, and scanning. The upper limbs were not wasted and showed some small white scars, which the patient now confessed were caused by syphilis. The slightest voluntary movement of either upper limb produced marked intention tremor. Every few minutes he had involuntary jerkings of one or the other upper limb. The biceps tendon reflex and the triceps tendon reflex were lost on each side. Sensation to touch and pain seemed to be unaltered in the upper limbs. There was no wasting of the hands or of the trunk muscles. The grasp of each hand was feeble. The lower limbs were not muscular, but there was no localized atrophy. The patellar reflex was completely lost on each side, even on reinforcement. The lower limbs could be moved in all parts, but the movements were feeble and intention tremor was pronounced in most of them. The tendo Achillis jerk was lost on each side. Babinski's sign was not distinctly present on either side, the movements of the toes being uncertain. Sensation for pain was normal in the lower limbs, but his answers regarding tactile sensation were unreliable because of his mental dulness. If not aroused he had a tendency to fall into a slightly stuporous state.

March 23. Mental aberration was present; he was delusional and swore considerable.

April 10th. He was somewhat quieter. Pulse rapid, but of good tension; temperature 101°; respirations increased in frequency and shallow. Examination of the chest was negative. He died on April 16, at 8 P.M.

At necropsy the brain and spinal cord appeared normal. They were removed and preserved in 10 per cent. formalin solution for histological examination.

In sections from the lumbar region of the spinal cord stained by the Weigert hematoxylin method, the posterior columns appeared much degenerated in the manner usually seen in tabes, the cornu-commissural zone and the oval field of Flechsig escaping. The lateral columns in the region of the crossed pyramidal tracts were a little less deeply stained than the rest of the anterolateral columns. Sections stained by acid fuchsin and hemalum showed round-cell infiltration of considerable intensity in the pia, but not about the bloodvessels within the spinal cord. The bloodvessels in the pia and those in the anterior roots were much congested and thickened. The nerve cells in the anterior horns stained by the thionin method were in a fairly good state of preservation, but here and there a very highly diseased cell was found.

The round-cell infiltration of the pia was more pronounced in the thoracic region than in the lumbar cord, and there was also a slight cellular infiltration about some of the bloodvessels of the spinal cord, especially in the sclerotic areas in the posterior columns. The posterior columns, except the cornu-commissural zones and along a part of the posterior septum, appear intensely degenerated in sections stained by the Weigert hematoxylin method. The crossed pyramidal tracts were a little less deeply stained than the rest of the lateral columns. The columns of Lissauer were degenerated.

In the cervical region the posterior columns were still intensely degenerated, and the degeneration resembled that of tabes. The lateral columns appeared to be normal. Round-cell infiltration of the pia was about as intense as in the lumbar region, but there was no distinct infiltration about the bloodvessels in the spinal cord. The nerve cells of the anterior horns were in about the same condition as those in the lumbar region; here and there a nerve cell was seen that was much degenerated.

Sections of the medulla oblongata stained by the Weigert hematoxylin method appeared to be normal. The hemalum-acid-fuchsin stain showed the round-cell infiltration of the pia to be as intense here as in the spinal cord, but it was not present about the bloodvessels within the nervous substance. Sections through the pons and through the cerebral peduncles showed the same round-cell infiltration in the pia, but the sections appeared otherwise normal. The cells of the nuclei of the hypoglossal, facial, abducens, trochlear, and oculomotor nerves, as well as the nucleus ambiguus, appeared normal when stained by the thionin method.

The pia of the right and left paracentral lobules was infiltrated with round cells and there was a distinct round-cell infiltration about the bloodvessels within the cortex. The cells of Betz appeared to be in a very fair condition, although here and there a cell was found partly degenerated.

Round-cell infiltration about the optic chiasm was only of moderate intensity, and was also found within the chiasm about the bloodvessels. The optic nerves and chiasm stained well by the Weigert hematoxylin method.

Sections of the cerebellum showed a moderate round-cell infiltration of the pia mater. Some of the Purkinje cells showed peripheral displacement of the nucleus.

The oculomotor nerves appeared to be partially degenerated and presented considerable round-cell infiltration within the nerves and an overgrowth of the connective tissue. Both sixth nerves appeared partially degenerated, when stained by the Weigert hematoxylin method, and there was considerable overgrowth of connective tissue within them. The motor and sensory roots of the left fifth, the left seventh, the left ninth and tenth, and the right twelfth nerves appeared fairly normal by the hemalum-acid-fuchsin and Weigert hematoxylin stains.

In order to show the resemblance of this case to one of multiple sclerosis it may be well to give a brief summary of it.

A young man positively denying, during several years, syphilitic infection, presented marked ataxia of gait, intention tremor of the limbs, and, a month or two before death, of the muscles of the face; scanning speech; at first normal iridic reactions, later Argyll-Robertson pupils, with unequal pupils; pallor of the temporal side of the right optic nerve and simple atrophy of the left optic nerve; and vertigo. Pain was never a prominent symptom. Remissions did not occur during the years he was in the hospital. Nystagmus was not observed. Shortly before his death, when some scars on his limbs were discovered and a history of syphilis obtained, the diagnosis of cerebrospinal syphilis was entertained, although it was not forgotten that a patient with multiple sclerosis is not exempt from syphilis.

The lesions found were those of meningo-encephalomyelitis, consisting chiefly of round-cell infiltration and degeneration of the posterior columns of the cord.

The patient was married and was the father of a healthy child. He positively denied having had syphilis until a short time before his death; on the other hand, his occupation, in which he was constantly exposed to the weather, and a history of having had a severe exposure to cold a short time before the onset of his illness, furnished a possible etiology for multiple sclerosis. The influence of exposure to cold and damp in the causation of multiple sclerosis has been referred to by Krafft-Ebing, Spiller, and others. Pain was never a marked feature of the case, and at no time was of the sharp, shooting character usually associated with inflammatory lesions of the meninges involving the posterior roots. The attacks of vertigo, as indicated by the repeated falls from his sweeping machine, are also in favor of the diagnosis of multiple sclerosis.

The gait in many cases of spinal syphilis, as also in multiple sclerosis, is of the spastic-paretic type, but the ataxic type is common. In this case there was marked ataxia in gait and station, which, however, was not increased by closing the eyes, so that it resembled the incoördination of gait characteristic of multiple sclerosis. The tremor was of the so-called intention type, was widely distributed, and involved, late, the tongue and the muscles of the face as well as the limbs. This intention tremor is regarded generally as being diagnostic of multiple sclerosis. The speech was "scanning," but there was no nystagmus. Uhthoff[2] found nystagmus present in only 58 per cent. of cases of multiple sclerosis, so that its absence in this case was not regarded as particularly significant. In six cases observed by E. W. Taylor,[3] in which the diagnosis was confirmed by necropsy, nystagmus seems to have been observed in only one or two, and then only on lateral deviation of the eyeballs. Uhthoff calls attention to the analogies between the ocular changes that occur in cerebrospinal syphilis and those of multiple sclerosis; the frequency of visual changes without ophthalmoscopic findings and recovery from them, the unilaterality of the symptoms, with the great infrequency of double total blindness. The changes in multiple sclerosis are of a less severe grade than those of syphilis; hemianopsia is rare; ophthalmoscopic changes are less marked and may last longer without alteration of vision. Optic neuritis is relatively seldom found in multiple sclerosis. A simple atrophy and pallor occur in both diseases. Parinaud[4] calls attention to the fact that paresis of a single eye muscle ending in recovery is frequent in multiple sclerosis, while complete or lasting paralysis rarely occurs, which it does, however, in syphilis. The findings in our case of a simple atrophy of one optic nerve and an atrophy limited to the temporal half of the other were very suggestive of multiple sclerosis.

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[2] Archiv f. Ophth., Band xxxix, Heft 1 und 3.

[3] Journal of Nervous and Mental Disease, June, 1906.

[4] Progrès méd., August, 1884.

The knee-jerks and the Achilles jerks are usually exaggerated in both multiple sclerosis and cerebrospinal syphilis, but this symptom depends on the location of the foci of the disease, and as has been shown by one of us (Spiller[5]), the knee-jerks may be lost in multiple sclerosis if the sclerotic foci invade the reflex arcs.

Summing up the symptoms in this case, it is evident that there was enough or more than enough to make the diagnosis of multiple sclerosis, and that there were no symptoms that were incompatible with such a diagnosis. Paoli Pini,[6] reporting cases from Oppenheim's clinic, does not hesitate to make the diagnosis of multiple sclerosis on far less evidence. The literature contains a number of papers devoted to the resemblance of cerebrospinal syphilis to multiple sclerosis, but most of the reported cases are without confirmation by necropsy. B. Sachs[7] pointed out the close resemblance between multiple sclerosis and cerebrospinal syphilis, and remarked that spastic paraplegia of the lower extremities, ocular palsies, optic-nerve atrophy, loss of pupillary reflexes, difficulties of speech, mental changes, apoplectiform seizures, marked remissions in all the symptoms are common to both affections. Nystagmus, intention tremor, and scanning speech are characteristic of multiple sclerosis, and are supposed to be absent in syphilis of the nervous system, but he has seen nystagmus and peculiarities of articulation resembling scanning speech in syphilis. In some cases of syphilis a tremor occurs which differs from the ordinary intention tremor only in that it is persistent and is aggravated on voluntary movements.

Sachs emphasizes the importance of the iridic reflex in making a diagnosis between multiple sclerosis and cerebrospinal syphilis, and considers the complete immobility of one or both pupils strong evidence in favor of syphilis rather than of multiple sclerosis. Indeed, in one of Cassirer's cases of doubtful diagnosis Sachs says that he (Sachs) did not regard the case as one of syphilis because the pupillary reflexes remained normal. In our case of syphilis pupillary rigidity was not present; at first the reflexes were normal; two years later the Argyll-Robertson pupil was observed. Nonne,[8] in his monograph on syphilis of the nervous system, says, in regard to the relation of syphilis to multiple sclerosis, that the latter cannot be mistaken for the former when the clinical picture of multiple sclerosis, as drawn by Charcot, is considered. The course of syphilis of the nervous system is different, and scanning speech, rotatory nystagmus, intention tremor, the general tremor of the trunk and head are not observed in spinal syphilis; this statement can no longer be accepted. If a patient with multiple sclerosis has a history of syphilitic infection the latter proves nothing, and it is probable that syphilis plays no part in the etiology of multiple sclerosis.

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[5] Amer. Jour. Med. Sci., January, 1903.

[6] Deut. Ztschr. f. Nervenheilk., vol. xxiii, p. 267.

[7] New York Med. Jour., 1891; Phila. Med. Jour., 1898.

[8] Syphilis und Nervensystem, S. Karger, Berlin, 1902, p. 306.

Oppenheim[9] thinks that the symptoms of cerebrospinal syphilis may closely resemble those of multiple sclerosis, especially in cases with spastic paraplegia. In both diseases there are cerebral and spinal symptoms, and in each remissions and exacerbations occur. Disturbance of vision, anesthesia, paresthesia, etc., may vary in intensity in each. However, it is rare for the diagnosis to be difficult for a long period, and yet it was difficult in our case for four years. The cerebral symptoms of multiple sclerosis, he says, are very different, viz., nystagmus and scanning speech; and the tremor is almost pathognomonic; stupor does not occur in multiple sclerosis except in the rare apoplectiform attacks, and is almost always observed at some period or other in cerebrospinal syphilis; but it was not present in our patient until a month or two before death. Oppenheim confesses that he has not always been able to decide whether a certain case was one of cerebrospinal syphilis or multiple sclerosis. The partial optic atrophy is the typical form of disease of the optic nerve in multiple sclerosis; when it occurs in syphilis it is usually accompanied by much impairment of sight, whereas an equal degree of atrophy in multiple sclerosis impairs the sight very little. In our patient, in 1903, vision was $\frac{5}{20}$ in the right eye and $\frac{5}{15}$ in the left. Pupillary symptoms are rare in multiple sclerosis.

The spinal symptoms may be very similar in the two diseases. It is strange, he says, that authors have paid so little attention to the differential diagnosis between cerebrospinal syphilis and multiple sclerosis. He refers, however, to the writings of Sachs, Sanger, Gilbert and Lion, Schuster, Uhthoff, Reinhold, Sottas, Marie, v. Bechterew, Cassirer, and Pini, and to these should be added those of Gowers and Krewer.

Some authors hold that syphilis may cause areas of disseminated sclerosis, but does not produce exactly the same symptom-complex as does the true multiple sclerosis. This subject is discussed recently by Guinio Catola.[10] He refers to a case reported by Schuster,[11] in which symptoms of disseminated sclerosis were associated with syphilitic ulceration of the pharynx, indurated glands, and periostitis of the right tibia. All symptoms disappeared under antisyphilitic treatment. This case probably was similar to the one we describe, as one could hardly expect sclerotic areas to disappear under any treatment, and it shows in association with our case that the symptoms of disseminated sclerosis may be caused by the syphilitic infiltration and vascular lesions. The symptoms in Schuster's case were intention tremor of the upper and lower limbs, dragging of the feet, exaggeration of the tendon reflexes, slight contractures of the limbs, and true nystagmus. Catola reports two cases with necropsy which he thinks bear on the question of the syphilitic origin of multiple sclerosis. Müller,[12] in his recent book on multiple sclerosis, refers to the resemblance this disease may have to cerebrospinal syphilis, but draws his conclusions from the literature rather than from personal experience.

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[9] Die syphilitischen Erkrankungen des Gehirns, 1903, 2d ed., p. 5.

[10] Nouvelle Iconographie de la Salpêtrière, July and August, 1906, p. 337.

[11] Berl. klin. Woch., 1885.

The difficulties in the diagnosis have been regarded as the cause of the apparent comparative rarity of multiple sclerosis in America. It may be that many cases, especially those of the *formes frustes*, are overlooked by superficial examination, but, on the other hand, there seems at present a danger that much will be called multiple sclerosis that in reality is some other disease.

RECURRENT FACIAL PALSY, WITH REFERENCE TO CERTAIN ETIOLOGICAL FACTORS.

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Oppenheim classes facial palsy as "the most common neural paralysis." Charles K. Mills speaks of it as "one of the most frequent and therefore one of the most important forms of paralysis." Allen Starr gives 7 per cent. as the number of facial palsies that recur on the same or the opposite side. Wm. G. Spiller believes this to be a fair estimate. I wish to present the records of two cases of the recurrent type that were treated at the Dispensary for Nervous and Mental Diseases of the Polyclinic Hospital, and I take this opportunity to thank Dr. Spiller for his permission to report them. In addition to the records of these two cases I shall briefly discuss an underlying cause predisposing to facial palsy in general that, I believe, has not been emphasized sufficiently.

Case I.—Jacob S., a tailor, aged forty-six years, was born in Russia. Both of his parents were over sixty years of age when they died. One brother died of tuberculosis. Four brothers and one sister are alive and apparently healthy. He denies venereal disease and says he has always been healthy. There are three other items of importance in his history: (*a*) He has been for years an habitual user of alcoholics in moderation and cigarettes in great excess; (*b*) he eats a great deal of meat; (*c*) he has had a number of attacks of painful throat that were not inflammatory in character—they were rheumatic attacks, to judge from his description of them. He came to the

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[12] Die multiple Sklerose des Gehirns und Rückenmarks, p. 250.

Polyclinic Hospital October 21, 1904, complaining of left-sided facial palsy associated with pain in the same side. The aural examination was made by Dr. Packard, who reported a normal condition. The notes read: "He cannot move, on the affected side, the muscles of his forehead, face, or mouth. The mouth is drawn to the right on innervation. The nasolabial fold is absent on the left side and the left eye cannot be closed. He can bite equally well on both sides and his tongue is protruded in the median line. The reactions to the faradic and galvanic current are normal."

Ten days later his general condition was as before, but the electric reactions were lost. Eventually this case resulted in a cure. It was a typical case of the classical type, without any special cause assigned to it.

On June 12, 1905, while riding in a train he felt a recurrence of the symptoms of palsy on the *right* side of his face; it soon became complete as before. He is positive that the window next him was not open and asserts that three days before this (June 9) he had felt decided pain behind the right ear. The duration of this second attack was shorter and the course less severe than the first, and on the tenth day the notes report a prompt reaction to electric stimulation of nerve and muscle.

Case II.—Jacob K. is four years old and was born in Philadelphia. His parents are alive and generally well; his mother complains a good deal of rheumatic pains in her muscles. There are six children in the family. One child, aged thirteen years, has, according to the mother, disease of the kidneys; the rest, including the patient, have always been considered healthy. His mother says the patient had an attack similar to the present one two years ago. The history of the second attack is that he woke on the morning of June 3, 1905, with the symptoms of left-sided facial palsy—the same side that was affected by the first attack. He had been sleeping in a draught during the night of onset. There was no pain or tenderness associated with this attack. Physical examination showed complete paralysis of the left side of the face, somewhat exaggerated knee-jerks and normal electric reactions over the affected area. Sixteen days after this the functions of the affected side were to all appearance normal and the reaction of nerve and muscle equal on both sides excepting the lips.

Various etiological factors have been advanced to explain this "commonest neural paralysis."

Oppenheim asserts: "There is no doubt that exposure to cold (draughts, sleeping beside an open window, etc.) is the cause of 70 per cent. of the cases in otherwise normal individuals." And, as a matter of fact, these are the cases that are seen so often in the nervous dispensaries—patients who are otherwise normal, apparently. Their histories do, in the great majority of cases, include an exposure to cold as the given cause of the attack; and yet to all appearances they are perfectly normal individuals and lack the special causes that would explain the attack, such as petrosal disease, suppurative conditions in the region of the facial nerve, etc. Indeed, they often seem to lack any sufficient cause or sum of causes that would account for their condition. The natural query is, Why, then, should these people be subjected to this alarming set of symptoms?

Answers to this have been many and varied. Of those who see a great many of these cases, a number believe that in very many of them the predisposing cause is a congenital or acquired narrowing of the bony canal through which the facial nerve makes its way. This could certainly explain why a number of these otherwise normal people, given some exciting cause, would be liable to this form of paralysis. But in so few cases can this be demonstrated that, while it explains the liability, it is not altogether satisfactory as regards the first cause.

There is one simple cause that I believe has not been emphasized sufficiently, and that is a defective condition of the intestinal tract associated with a faulty elimination of waste products by way of the kidneys, the lowered tone of the nerve being a secondary and consequent symptom.

We know the class of persons (both overfed and underfed) who complain of neuralgias, rheumatic pains, gouty affections, etc. Some of them are distinctly neuropathic, others are not and yet they are subject to the same ailments, and so many of them present themselves suffering from the alarm caused by a facial palsy and can give no satisfactory cause. In attempting to find an explanation for their attacks my attention was forcibly drawn to the fact that most of those I saw presented a coated tongue, told of former headaches or neuralgias, or complained of rheumatism or attacks of rheumatic sore throat. As to sore throat Dr. Grayson asserts that 9 out of 10 cases of sore throat, tonsillitis, and painful throat originate in gastro-intestinal disorder and are allied to gout. We know how often the common neuralgic pains and rheumatic pains are relieved together with the relief of the liver and bowels; and how many are helped when we have discovered that the urine passed in twenty-four hours amounts to a good deal less than the normal and correct this condition of faulty elimination.

The medicinal treatment has been to a certain extent empirical, but is the same as we prescribe for rheumatism, gouty affections, sore throat, and the like. It is well known that the salicylates are exhibited with good results in these cases of facial palsy. The same remedy associated with catharsis is indicated in the other list. Brunton asserts that the salicylates are carried as such in the blood and their antiseptic qualities are well known, and he recommends them together with the bromides in gouty and lithemic cases for their nervous irritability. He further asserts in this connection that "no other drug causes such a flow of bile."

There appears to be a true basis for the belief that these varying ailments are in some way closely connected.

I believe that these cases of facial palsy are associated with the diseases that are brought about by a storing up in the body of the toxins or poisons normally thrown off by the bowel, skin, and kidneys, and that they are due to a constant or repeated poisoning of the nerves by a circulation that is vitiated by this unhygienic state of affairs. The exciting cause may be exposure, traumatism, or an added intoxication, and yet the result is due to the underlying condition of the nerves themselves, which in turn is due to the faulty elimination.

Applying this to the two cases in hand as being typical facial palsies, we recollect that Case I—the man—is a tailor (an occupation notorious for gastro-intestinal disorders), who imbibed alcoholics regularly, ate meat in excess, and used tobacco to excess. He had a thick, coated tongue and an offensive breath. His history showed repeated attacks of sore throat and he had frequent headaches.

In such a man, saturated with the intoxication from three sources and given, let us say, a nerve constricted and engorged and situated in the most exposed part of the body; now let that man ride in a draughty car in summer, thus aiding the rapid evaporation of the perspiration that would naturally flow excessively, and there is an ideal situation for the onset of a facial palsy.

In Case II we have a boy of a neuropathic habit, another one like Case I, of that class of people that are so wrongly fed and hard worked—the Russian Jews—and showing, besides the paralysis, no other symptoms than a coated tongue, lack of nourishment due to gastro-intestinal inaptitude, and having a mother afflicted with neuralgias most of the time. The parents of the boy were well-fed individuals and said they tried to overfeed the boy, hoping to fatten him.

Both of these cases can better be explained by the lack of proper alimentary hygiene than in any other way. Of course the direct or exciting cause is often as in Case II, exposure to draughts and cold while the natural body resistance is lowered—as during sleep or in the summer or when the perspiration is too quickly evaporated over exposed skin surfaces, etc.—but most often the underlying cause is, I believe, disordered hygiene of the alimentary tract and the consequent failure to eliminate waste products through the natural avenues. The good effect of eliminative and antiseptic treatment, with proper hygiene of the alimentary tract, supports this belief.

THE BACTERIOLOGY OF THE BLOOD IN TYPHOID FEVER.

AN ANALYSIS OF 1602 CASES.[1]

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During the last six years we have made bacteriological examinations of the blood in 123 cases of typhoid fever in the wards of the Second Medical Division of Bellevue Hospital. In 1904 we published an analysis of 604 cases of typhoid fever (for the most part collected from the literature) in which the blood had been examined bacteriologically. Seventy-five per cent. of these cases, examined at all stages of the disease, showed the presence of the typhoid bacillus. Bacteriological examinations of the blood of typhoid and suspected cases have been made routine practice in many hospitals, and the number of cases reported to date, including our own, reaches a total of 1602. The present analysis includes all the cases in our former paper, except the reports of individual cases.

Methods. We usually draw 10 c.c. of blood into an all-glass syringe from a vein at the bend of the elbow. In our earlier experiments (1901 to 1903) we used broth flasks, putting 2 to 3 c.c. of blood into each 100 c.c. of broth. Later, on learning that Busquet and other French authors had had extraordinary success by using very large quantities of broth, we followed their method of diluting each cubic centimeter of blood in about 200 c.c. of broth, but the results were not appreciably better than before. Since August, 1906, however, we have had very marked success with ox-bile, as recommended by Conradi. Ox-bile not only prevents coagulation, but inhibits the bactericidal action of drawn blood and affords an excellent culture medium for typhoid bacilli. Our tests on this point have fully confirmed the previous observations of Conradi, Kayser, and others.

The method followed has been to take ox-bile 90 c.c., glycerin 10 c.c., and peptone 2 grams. The mixture is distributed into small flasks, 20 c.c. in each, and sterilized. Three of these flasks are used for each examination, about 3 c.c. of blood being run into each. The flasks are then incubated, and the next morning streaks are made from each flask over the surface of a litmus-lactose-agar plate. If microörganisms are present, a growth may be observed in five or six hours. If the growth does not redden the medium and is found to be a bacillus resembling the typhoid organism, it is tested for the Widal reaction with immune serum. By this procedure we are often able to determine if the case is one of typhoid fever or not within twenty-four hours after drawing the blood.

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[1] Read at a meeting of the New York Academy of Medicine, March 7, 1907.

We give below a table of the cases included in this report:

TABLE OF REPORTED CASES.

Number.	Authors.	Number of cases.	Bacillus found.	Bacillus not found.
1	Ruediger	32	20	12
2	Fox	14	1	13
3	Cole	15	11	4
4	Hewlett	25	21	4
5	Widal	25	17	8
6	Busquet	83	83	0
7	Courmont and Lesieur . . .	37	37	0
8	Schottmueller	119	98	21
9	Kerr and Harris	56	31	25
10	Castellani	14	11	3
11	Auerbach and Unger . . .	10	7	3
12	Kuehnau	21	9	12
13	Warfield	48	33	15
14	Janesco	19	8	11
15	Rolly	50	43	7
16	Berri	13	7	6
17	Duffy	88	62	26
18	Hirsh	97	75	22
19	Memmi	30	18	12
20	Richard	50	44	6
21	Perquis	40	38	2
22	Fornet	19	14	5
23	Jochmann	30	25	5
24	Harrison	8	5	3
25	Eppenstein and Korte . . .	6	5	1
26	Ruáta	12	12	0
27	Korte and Sternberg . . .	22	22	0
28	Trappe	38	25	13
29	Brion and Kayser	233	153	80
30	Kayser	75	50	25
31	Loiseleur	65	63	2
32	Epstein	85	68	17
33	Coleman and Buxton . . .	123	81	42
		1602	1197	405

The table shows that of the total of 1602 cases, 1197 (or 75 per cent.) gave a positive result. The examinations were made at all stages of the disease and by different methods. Since in our experience the bile method[2] is the only one which may confidently be depended upon, such a large percentage of positive results goes far to prove that the bacillus is present in the blood in practically all cases of typhoid fever.

ANALYSIS OF THE CASES BY WEEKS. The day of the disease upon which the examination was made is mentioned in 1137 cases only. To be more exact, this represents the number of examinations, not of cases, for in many instances more than one examination was made in a case.

First Week. Of 224 examinations in the first week of the disease, 200 (89 per cent.) were positive. The earliest positive result has been reported by Widal, who recovered the bacillus from the blood on the second day of the disease. The reported positive results become more frequent as the end of the first week is approached, only, we believe, because the disease is not suspected earlier and the examinations made, or because the cases do not come under observation.

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[2] We have not tried the glucose method of Epstein.

Second Week. Of 484 examinations made in the second week of the disease, 353 (73 per cent.) were positive.

Third Week. Of 268 examinations made in the third week of the disease, 178 (60 per cent.) were positive.

Fourth Week. Of 103 examinations made in the fourth week of the disease, 39 (38 per cent.) were positive.

After the Fourth Week. Of 58 examinations made after the fourth week of the disease, exclusive of relapses, 15 (26 per cent.) were positive.

Very few statements are made concerning the clinical histories of the cases in and after the fourth week, though some of them are reported as severe and of long duration. As in our first analysis, the percentage of positive results is greatest in the first week and steadily declines thereafter.

We have already called attention to the remarkably successful results of Busquet and others, who recovered the bacillus from the blood in approximately 100 per cent. of their cases. Our results since the adoption of the bile method have been equally successful, standing out in marked contrast to those with broth. In all we have used this method in 34 cases. As a rule, the blood was examined as soon as a case of fever without obvious cause entered the hospital, and before the diagnosis was established. Six of these 34 cases were diagnosticated ultimately as certainly not typhoid fever; 3 of the cases pursued the clinical course of typhoid fever, but gave neither a positive blood culture nor a positive serum reaction against any member of the typhoid-colon group. In fact, after examinations of urine, feces, opsonic index, and injections of tuberculin, a satisfactory diagnosis could not be made. It seems only fair to exclude these cases. One case ran an eleven-day temperature, the maximum being 101°, but for the most part ranging between 99° and 100°. The first blood culture was taken on the eighth day. There was a difference of opinion as to the diagnosis. The patient had an old tuberculous process at one apex. This case likewise may be fairly excluded. We made three examinations of his blood.

The remaining 24 cases were typhoid fever clinically and by serum reaction and all gave positive bacteriological results. The examinations were made from the fifth to the twenty-first day in a long-duration case.

The various series of cases in the table giving approximately 100 per cent. of positive bacteriological results are too numerous to be accidental. They compel the conclusion that the typhoid bacillus is present in the blood in every case of typhoid fever and that failure to recover it is due to error of technique. The diminishing percentages of the larger analysis in the later weeks of the disease do not indicate, then, that the bacillus has disappeared from the blood in the negative cases, but point rather to diminishing numbers of bacilli, whose presence imperfect methods have failed to reveal. All investigators except Conradi are agreed that the bacillus disappears from the blood at or about the time the temperature falls to normal. Conradi claims that the bacillemia persists into convalescence. We have repeatedly examined the blood in the last day or two of the febrile period and not once have we recovered the bacillus. Therefore, it seems probable that the typhoid bacillus is not only present in the blood in every case of typhoid fever, but that it is present throughout the course of the disease, or at least to within a day or two of complete defervescence.

THE SIGNIFICANCE OF THE BACILLEMIA. If future observations confirm the conclusion that the typhoid bacillus is present in the blood of every case of typhoid fever throughout its course, the current conception of the pathogenesis of the disease should be modified. Typhoid fever can no longer be regarded simply as an infection of the body with typhoid and related bacilli (Bacillus paratyphosus, etc.). The typhoid bacillus may be present in the body and actively growing, yet the patient not have typhoid fever. It has been shown, for example, that the bacillus may live and multiply in the intestine of healthy persons. The patient is infested and a menace to others, but is not infected. The number of cases of biliary infection with the typhoid bacillus, without a previous or existent typhoid fever, is fairly large and is increasing. At least two cases of cystitis, caused by the typhoid bacillus in persons without a history of typhoid fever, have been recorded. (There is little probability, however, of the absorption of endotoxins in any quantity in cholecystitis and cystitis). In the post-typhoid bone and other inflammatory lesions the lodgement and growth of the bacillus do not produce the characteristic symptoms of typhoid fever, in spite of the fact that large amounts of endotoxin should be liberated and absorbed when the abscesses are multiple. The very term used to describe these conditions, "post-typhoid," indicates that the typhoid fever *per se* has subsided. The temperature curve conforms to the so-called septic type.

Therefore, it seems that to produce typhoid fever the bacillus must not only be present in the body and growing, but that it should grow in a situation whence it has free access to the blood. In our first paper we expressed the opinion that in typhoid fever the earliest and principal seat of infection is the blood, and that the disease should be regarded as a bacillemia. From the work done by one of us on the absorption of the typhoid bacillus from the peritoneum, and from the fact that in typhoid fever the lymph nodes and spleen contain such enormous numbers of bacilli, we are disposed to modify this view and to conclude that in typhoid fever the bacillus first finds its way from the alimentary tract to the lymphopoietic system, including the spleen, where it develops chiefly and from which it invades the blood stream. We think it doubtful whether the bacillus multiplies in the blood, but rather that its presence there represents simply an overflow from the lymph organs. Under this interpretation the presence of the bacillus in the blood does not constitute a true septicemia.

The absorption experiments above referred to also indicate that destruction of the typhoid bacilli proceeds most rapidly in the blood. This observation, together with the fact that the bacillemia persists throughout the disease, suggests the following view of the pathogenesis of typhoid fever: That the disease is caused by the destruction of vast numbers of bacilli in the blood, with the liberation of their endotoxins, and the consequent reaction on the part of the host. When the endotoxins are liberated elsewhere in the body, *e. g.*, in abscesses, the symptomatology is not that of typhoid fever.

This conception of the nature of typhoid fever is borne out by analogy. It is known that Bacillus paratyphosus may infect the intestine and produce the clinical picture of gastro-enteritis, but that when it invades the lymph organs and blood it produces a disease clinically indistinguishable from typhoid fever. Diplococcus lanceolatus and the various streptococci furnish similar analogies in that they produce different affections according to the regions they attack.

There is another matter to which we would call attention in this connection. The idea still prevails in some quarters that the course of typhoid fever may be influenced and even shortened by the use of intestinal antiseptics. Such opinion is based on an erroneous conception of the nature of typhoid fever. After invasion of the body proper by the bacillus the battle-ground shifts from the intestines to the blood, and the employment of intestinal antiseptics with the idea of controlling the disease is, to say the least, irrational.

THE RELATION OF THE BACILLEMIA TO THE COURSE AND TYPES OF THE DISEASE. *Course.* The analysis of the cases in the various weeks of the disease suggests the following relation of the bacillemia to the course of typhoid fever: In the earlier stages the bacillus invades the blood in greatest numbers. Later, as the disease is approaching a favorable termination, the diminution in the number of bacilli in the blood is simply an index of less active development in the lymphatics and spleen. If the disease in any case pursues a long-duration course, that beyond the usual three weeks, the bacillus may be recovered from the blood as long as the temperature persists. We have isolated the bacillus late in such cases repeatedly. There appears then to be a definite relation in the evolution of typhoid fever between the symptoms and the bacillemia. The increasing intensity of the symptoms in the earlier period of the disease corresponds to active growth of the bacilli. They invade the blood stream in increasing numbers and are there destroyed. Then comes the stationary period, when the ratios of growth and destruction appear uniform. The steep-curve period corresponds to a diminishing bacillemia, and defervescence to the complete disappearance of bacilli from the blood. In other words, the duration of the febrile movement is measured by the persistence of the bacillemia. As already stated, Conradi is the only investigator who claims that the bacillemia continues into convalescence.

Ewing has expressed the opinion that "the degenerative changes in the liver, kidneys, and lymphoid organs, while initiated by the bacterial proteids, possess certain self-perpetuating tendencies," and therefore "typhoid fever is a combination of a specific bacterial intoxication and a somewhat peculiar auto-intoxication, the former element being more prominent early, the other later in the disease, but both developing simultaneously." We are unable to accept this conception of the pathogenesis of typhoid fever. It appears inconsistent with the facts developed by our studies. We maintain that exclusive of convalescence, which should be regarded as the period of repair, degenerative changes occur only in the presence of active growth and destruction of bacilli. We have shown that convalescence is established immediately upon the disappearance of the bacilli from the blood, and there are reasons to believe that it is not interrupted except as the result of a fresh growth of bacilli. While the bacilli disappear from the blood at or just before defervescence, it is improbable that all the bacilli in the body have been destroyed. Otherwise, relapses and post-typhoid inflammatory lesions would be impossible. Unless then it can be shown that the symptoms of typhoid fever would persist after the complete destruction of all bacilli in the body, we think that Ewing's position is untenable.

Types. The bacillemia apparently bears no relation to the type or severity of the disease except in so far as regards numbers of bacilli. The bacillus is found in the blood equally, but not with the same persistence, in the mild as in the severe cases, and in the cases of short as well as of long duration. We have found the bacillus, for example, in cases lasting ten, thirteen, and fourteen days, and on the twenty-seventh day of a long-duration case. The importance of the definite establishment of the nature of these short-duration cases can scarcely be overestimated from the epidemiological standpoint. The serum reaction has done much to clear up their diagnosis, but the final proof has remained for the bacteriological examination of the blood. We make only a brief reference to these cases here, as we shall deal with them at length in the near future.

Relapses. The blood has been examined bacteriologically by various investigators in 33 relapses. The typhoid bacillus has been recovered in 30 (90 per cent.) of the cases. We suggested in 1904 that a relapse in typhoid fever is due to reinvasion of the blood by the bacillus. Reinvasion of the blood with destruction of the bacilli probably cause the symptoms of a relapse, but the underlying conditions which inaugurate active development of the bacilli after their growth has once been brought under control are unknown. We feel safe in asserting that a relapse is not due to reinfection with the typhoid bacillus from the intestine as the result of intestinal trauma brought about by dietary irregularities. We do not wish to intimate, however, that we believe the occurrence of a relapse is entirely independent of diet or to be understood as advocating a liberal diet in typhoid fever. We are not prepared as yet to express an opinion upon this subject.

THE RELATION OF THE BACILLEMIA TO THE SERUM REACTION. In our former paper we stated that it would seem likely on *a priori* grounds that the typhoid bacillus is always present in the blood before the serum reaction develops, for the reason that endotoxins should be liberated before the agglutinins could be formed. The following table clearly illustrates the truth of this conclusion:

TABLE SHOWING RELATION OF BACILLEMIA TO SERUM REACTION.

Author.	Number of cases.	Bacillus found and Widal reaction negative.
Hirsh	100	23
Jochmann	30	5
Rolly	50	16
Duffy	88	18
Buxton and Coleman	123	32
	391	94

Of the 55 cases of Hirsh and ourselves, which showed the presence of the bacillus in the blood before the serum reaction could be obtained, 23 were in the first week, 26 in the second, and 6 in the third. The diagnostic value of these results in cases in which only one, or a few, serum tests have been made is important. A negative serum reaction may have no significance even in the third week of the disease. Moreover, Dr. Hastings has shown in some of our cases, especially those of short duration, that a positive serum reaction may be present for only two or three days. If the serum reaction had not been tested on those days, the result would have been recorded as negative throughout the disease. For the complete diagnosis of an obscure case by the serum reaction the tests should be made daily.

CONCLUSIONS. 1. The typhoid bacillus is present in the blood of every case of typhoid fever throughout its course.

2. The bacillemia in typhoid fever does not constitute a true septicemia, but it represents an overflow of bacilli from the lymphopoietic organs.

3. The clinical picture of typhoid fever results only from infection of the lymphopoietic organs by the typhoid bacillus, with invasion of the blood stream and destruction there of vast numbers of bacilli.

4. The endotoxins of the typhoid bacillus are not cumulative in action and convalescence from the typhoid fever *per se* is established within a few days after the disappearance of the bacilli from the blood.

We wish to express our thanks to Profs. Loomis, Thompson, and Dana for the privilege of studying the cases.

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INFLUENCE OF IODINE PREPARATIONS ON THE VASCULAR LESIONS PRODUCED BY ADRENALIN.

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ABOUT two years ago L. Loeb and T. G. Githens[1] instituted a series of experiments to determine the experimental conditions in which adrenalin injections into rabbits would cause arterial changes.

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[1] The Effect of Experimental Conditions on the Vascular Lesions Produced by Adrenalin, AMER. JOUR. MED. SCI., 1905.

The results obtained were as follows: (1) Pyrocatechin, even though injected in doses sufficiently large to produce convulsions and continued over a long period, produces no, or only trifling, changes in the bloodvessels, this in spite of the facts that pyrocatechin raises the blood pressure and that adrenalin and pyrocatechin bear to one another a distinct chemical relationship. It seems, therefore, that adrenalin does not act, or does not alone act, through its power of raising blood pressure in the production of the changes in the vessel walls. (2) Previous thyroidectomy does not hinder the action of adrenalin on the vessel walls, a conclusion contrary to that of Lortat-Jacob and Sabarianu, who have reported such an action. (3) The usual vascular changes do not seem to be influenced by simultaneous administration of potassium chromate and adrenalin, or by tying a ureter and then administering adrenalin, although potassium chromate was given in doses sufficiently large to cause albuminuria, during the whole course of the experiment. Thus the vascular lesions produced by adrenalin do not seem to be increased or decreased by experimental lesions of the kidney. (4) If pregnant animals were injected with adrenalin from the beginning of pregnancy the pregnancy followed its normal course, as did the labor; the aortas of the newborn showed no lesions, while those of the mothers showed less tendency toward change than did aortas of non-pregnant animals; at least the conclusion may be drawn that pregnancy does not predispose toward vascular changes. (5) The number of the injections, and the quantity used at each injection of adrenalin are of far less importance than is the length of time between the commencement of the injection and the death of the animal; this does not set aside the vascular changes which can be found after a short period, for several such cases were found. (6) Histologically there was primarily found a necrosis of the musculature of the media, the elastica being at first preserved. After this there follows a calcification, stretching of the elastica lamellæ, and then a splitting up and disappearance of the elastic membranes. These conclusions were supported by experiments on forty rabbits.

In their communication Loeb and Githens stated that this experimental method of producing lesions in bloodvessels will provide us with means of determining accurately the effect of certain substances believed to be curative of arteriosclerosis in man. Although there exists a marked difference between the histological characters of arteriosclerosis in man and the microscopic lesions produced by adrenalin injections, it is nevertheless not yet certain how fundamental these differences are and to what extent they may be determined by a special kind of response of the species used in the experiment; but even conceded that this difference between arteriosclerosis and changes produced by adrenalin is a decisive one, there exists in man a certain lesion of the bloodvessels of the extremities seen in old age in which the media alone is affected and in which marked calcification of this coat is found. Soon after the vascular changes produced in rabbits by the injection of adrenalin had been discovered, attention was drawn by some pathologists to the similarity between these vascular lesions in the extremities of old persons and the lesions produced by adrenalin. A certain similarity exists also between syphilitic aortitis and the lesions produced by adrenalin, as far as the localization of the lesions is concerned.

It is, therefore, not altogether without interest to test the effect of certain chemical substances on the vascular lesions in rabbits; under such experimental conditions we are able to determine, with much greater accuracy, the efficacy of these drugs, since we are able, at the same time, to conduct the necessary control experiments which we are rarely, or never, able to do in treating vascular lesions in man.

One of the first substances which suggested itself for experimentation was potassium iodide,[2] which lowers the viscosity of the blood and may in this way influence the circulation, counteracting the effect of the increased blood pressure and thus preventing changes of arterial walls. Shortly before we undertook these experiments a brief communication of v. Korangi[3] appeared in which he stated that by simultaneous injection of iodipin and adrenalin it was possible, almost entirely, to prevent the effects of adrenalin on the bloodvessels. Boveri[4] found a beneficial effect of iodipin in a few cases. Cummins and Stout[5] observed that in two rabbits simultaneously injected with adrenalin and potassium iodide, no vascular lesions were present after the death of the animal.

Quite recently, after our experiments had been almost concluded, there appeared a paper by J. Biland,[6] in which, in experiments carried on simultaneously and independently of those of Korangi, he came to the conclusion that potassium iodide far from having a beneficial effect, aggravated the lesions produced by adrenalin. Under these conditions it might not be without interest to give the results of our investigations, especially as they are based on a much larger number of experiments than those carried out by any of the aforementioned investigators.

In the first place we made (1) a comparative investigation of different iodine preparations, using iodipin as well as potassium iodide; (2) we applied the latter in some cases subcutaneously, in some cases intravenously, in order to determine whether the mode of application was of importance; (3) we used the various substances in different doses, using in some animals very large, in others very small doses; (4) we tested another substance, bromipin, in order to determine whether or not a possible effect of iodine preparations was a specific one; and (5) we included in our experiments the action of potassium sulphocyanide, because Pauli had suggested, from conclusions based on theoretical reasons, that potassium sulphocyanide might be able to take the place of potassium iodide in its therapeutic application and might even be more efficient than the iodine preparation.

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[2] The use of this substance had already been planned by Loeb and Githens in connection with their former experiments.

[3] Ueber die Wirkung des Iods auf die durch Adrenalin erzeugte Arterionekrose, Deutsch. med. Woch., April 26, 1906, pp. 679, 680.

[4] Deutsch. med. Woch., 1906, Nr. 22.

[5] University of Penna. Med. Bull., July, 1906.

[6] Deutsch. Arch. klin. Med., August 28, 1906.

Three series of experiments were carried out in the course of our work and the results are seen in the abstracts of our protocols.

In summarizing these results we shall divide each set into two classes: (1) Those animals which received but a few injections, and (2) those which received a comparatively large number of injections; we place those which received more than ten injections in the latter class, and all which died or were killed before receiving this number in the former class. In speaking of the changes in the aorta we will use four terms: *normal; changed* when there is unevenness but no true macroscopic calcification; *slightly calcified* when there are but few small plaques well scattered or only in one small locality; and *markedly calcified* when the calcareous plaques are large and cover a considerable portion of the vessel wall.

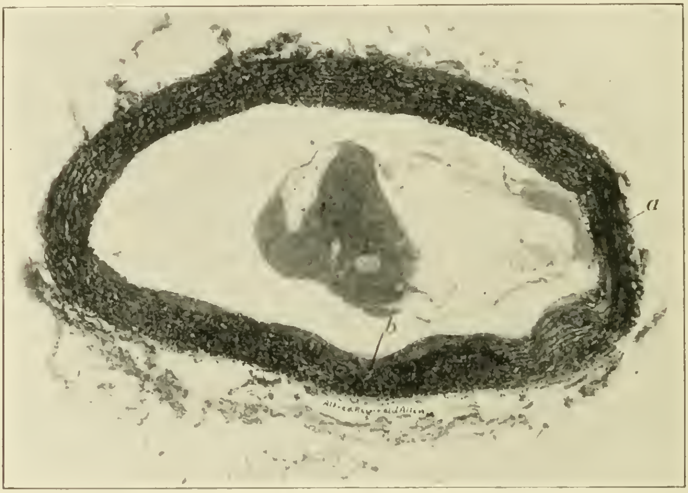

FIG. 1.—Section through one of the large vessels branching off from the arch of the aorta. In this case the aorta itself was normal macroscopically, although the smaller vessel shows calcareous plates (*a* and *b*). The rabbit had received 23 injections of adrenalin and iodipin (3.4 c.c adrenalin, 46 c.c. iodipin). It lived from July 8, 1906, until it was killed on August 22, 1906.

We find the results to be as follows:

SERIES I. *Set A* (*control*): Injected every other day; adrenalin 0.1, 0.15, 0.2 c.c. Short-lived: 3 rabbits, of which 2 showed normal aortas and 1 slight calcification. Long-lived: 8 rabbits, of which 1 was normal, 4 showed slight calcification of the aortas, and 3 showed marked calcification.

Set B (Iodipin and Adrenalin): Injected every other day; iodipin 2 c.c., adrenalin as in Set A. Long-lived: 4 rabbits; 1 presented a normal aorta, 2 were slightly calcified, and 1 showed marked calcification in the vessels. It may be worth noting the fact that in this series none of the rabbits died before the tenth injection.

Set C (KCNS and Adrenalin): Injected every other day; KCNS 0.1 c.c. of 5 per cent. solution, adrenalin as in Set *A*. Subcutaneously; short-lived: 3 rabbits; 2 showed a normal aorta, 1 showed a changed aorta. Long-lived: 2 rabbits; 1 showed the aorta normal, 1 showed slight calcification. Intravenously; short-lived: 2 rabbits; 1 showed a normal aorta, and the other showed marked calcification. Long-lived: 4 rabbits; 1 showed a normal aorta, 1 showed the aorta changed, 2 showed slight calcification. Those rabbits which received the KCNS subcutaneously died earlier and tended more toward diarrhœa than those receiving the injection intravenously. In this set KCNS was given in larger doses than in any of our later series.

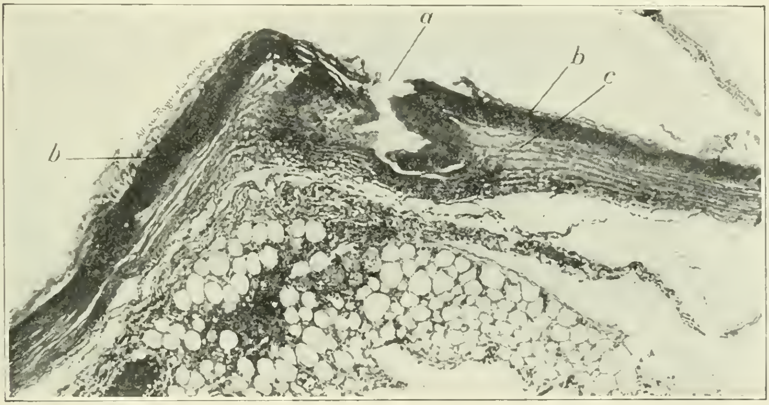

Fig. 2.—Section through the upper part of the descending aorta of the rabbit which had received 6 injections of adrenalin (0.8 c.c.), iodipin (18 c.c.), and KI (1.0 gram) in the course of twelve days. The aorta was much changed, showing calcification (*b*), necrosis, and distention (edema?) of the media (*c*), and a partial rupture of the wall of the aorta (*a*).

Set D (Bromipin and Adrenalin): Injected every other day; bromipin 2 c.c., adrenalin as in Set *A*. Short-lived: 10 rabbits; 4 showed a normal aorta, 4 showed simply a changed condition, and 2 had the aorta markedly calcified. Of the 4 which showed normal aortas, 3 died very early—before they had received more than four injections. Long-lived: 2 rabbits, both of which showed the aorta markedly calcified.

SERIES II. *Set H (control)*: Injected every day; adrenalin, 0.1, 0.2, 0.25, 0.3, 0.35, c.c. Short-lived: 1 rabbit which showed a normal aorta. Long-lived: 1 rabbit which showed a normal aorta.

Set E^1 (Iodipin and Adrenalin): Injected every other day; iodipin starting with 4 c.c. and running up the dose until 9 c.c. was given at each injection; adrenalin as in Set *H*. Short-lived: 3 rabbits; 1 showed changes in the aorta, 2 were markedly calcified. Long-lived: 3 rabbits; all 3 showed fairly marked calcification. In this set iodipin was given in the largest-sized doses used during the experiments.

Set E^2 (Iodipin, KI, and Adrenalin): Injected every other day; iodipin, 3 c.c.; KI 0.1, 0.15, 0.2 gram; adrenalin as in Set *H*. Short-lived: 6 rabbits; 1 showed the aorta changed, 2 showed slight calcification in the aorta, 3 showed the aorta markedly calcified. It is peculiar that all the animals of this series died shortly after the injections were started, none receiving more than seven injections, nor living more than fourteen days after the injections were begun. We noticed at the site of the KI injection (which was given subcutaneously) a hardening and blackening of the skin which gave the appearance of a charred piece of flesh; this condition was preceded by an intense inflammation which passed on to this blackened condition. There can be little doubt that the KI called forth this change.

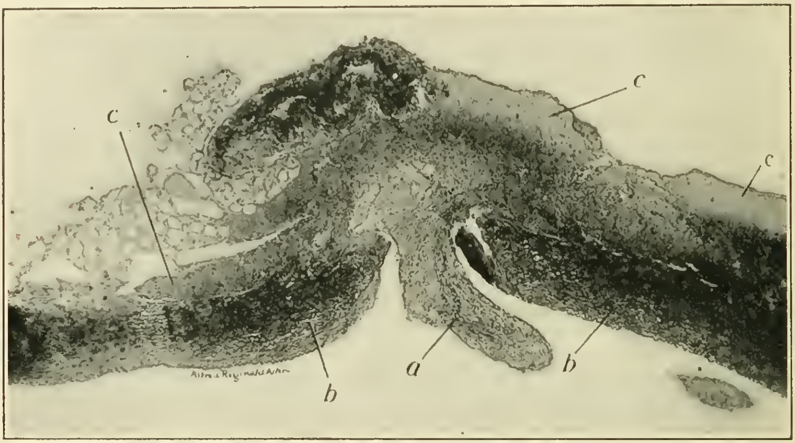

FIG. 3.—Section through the upper part of the descending aorta. The media of the vessel wall is entirely necrotic (*b*). The vessel had ruptured and a thrombus (*a*) formed. The extravasated blood (*c*) is found in the adventitia of the aorta. The rabbit had received 18 injections of adrenalin (2.4 c.c.) and iodipin (36 c.c.). It lived from July 8, 1906, until it was killed, August 11, 1906. The aorta was much changed.

Set G (KCNS and Adrenalin): Injected every other day; KCNS 0.025 c.c. (5 per cent. solution), adrenalin, as in Set *H*. Short-lived: 3 rabbits; all of which presented normal aortas. Long-lived: 3 rabbits; none showed any change in the aorta. In this set KCNS was given intravenously, as we believed from our results in our first series that it acted to better effect when administered in this fashion. Medium-sized doses of KCNS were used in this series.

Set F (*Bromipin and Adrenalin*): Injected every other day; bromipin, 0.5 c.c., adrenalin as in Set *H*. Short-lived: 3 rabbits; 1 showed a normal aorta, 2 showed the aorta markedly calcified. Long-lived: 3 rabbits; 1 showed a normal aorta, 1 showed slight calcification, and 1 showed marked calcification of the aorta.

SERIES III. *Set D* (*control*): Injected at first every other day, later every day; adrenalin, 0.1, 0.2, 0.3 c.c. Short-lived: 2 rabbits; 1 showed a normal aorta, 1 showed a slight change. Long-lived: 3 rabbits; 1 had a normal aorta, 1 showed changes, and 1 showed marked calcification. Here, as in the other control sets, we note the uncertainty of the action of adrenalin in producing vascular lesions.

Set A (*Iodipin and Adrenalin*): Injected at first every other day, later every day. Iodipin, 2 c.c.; adrenalin, as in Set *D*. Short-lived: 4 rabbits; 1 showed the aorta changed, 3 showed marked calcification.

Set C (*KCNS and Adrenalin*): Injected at first every other day, later every day; KCNS 0.012, 0.01 c.c. of (5 per cent. solution) adrenalin as in Set *D*. Short-lived: 2 rabbits; 1 showed the aorta normal, 1 presented marked calcification. Long-lived: 8 rabbits; 3 presented a normal aorta, 2 showed the aorta changed, 2 had slight and 1 marked calcification. KCNS, which was injected intravenously, was here used in the smallest doses given during the course of the experiments; the smallness of the dose may perhaps account for the increased effect of the adrenalin as compared with Set *G*, Series II.

Set B (*KI and Adrenalin*): Injected at first every other day, later every day; KI 0.015, 0.05, 0.058, 0.08 gram; adrenalin as in Set *D*. Short-lived: 1 rabbit, showing slight calcification in the aorta. Long-lived: 5 rabbits; 2 showed the aorta only changed, 1 showed slight calcification, and 2 showed marked calcification. KI was administered intravenously and in considerably smaller doses than in Set E^2, Series II.

We may summarize the results of these series as follows: of the 18 rabbits used in the control sets, 7 showed no changes in the aorta, 2 showed changes in the vascular walls, 4 showed slight calcification, and 4 showed marked calcification.

Of the 15 rabbits of the iodipin sets, only 2 showed normal aortas, 2 showed changes in the vessel, 2 showed slight, and 9 marked calcification of the vessel.

Of 18 rabbits injected with bromipin, there were 6 with normal aortas, 4 showed changes of the vessel walls, 2 showed slight calcification, and 7 showed marked calcification of the aorta.

Of 6 rabbits treated simultaneously with iodipin and KI none escaped the effects of the adrenalin; the aorta was changed in 1 case, 2 showed slight calcification, and 3 showed marked calcification.

Of 6 rabbits treated with KI, none was found with normal aorta, and 2 showed marked calcification of the aorta.

Of 27 rabbits treated with KCNS, 15 were found with normal aortas, 3 with the aorta changed, 6 with slight calcification, and 3 with marked calcification of the vessel walls.

It may be interesting to note here also the occurrence of ascites in these experiments; we found that 15 of the 90 rabbits on which autopsies were performed showed this condition. It appeared through all the various sets, appearing once in the control sets (Series III), once in the iodipin sets (Series I), once in the iodipin and potassium iodide sets (Series II), 6 times in the potassium sulphocyanide sets (Series I, and Series II), and 7 times in the bromipin sets (Series 1 and Series II).

Hemorrhagic gastritis occurred in 11 of the cases and of these, 6 were in rabbits treated with potassium sulphocyanide (Series I and Series II), 2 were in the control sets (Series I), 2 in the bromipin sets (Series I), and 1 in the iodipin set (Series I). The occurrence of gastritis seems to have some connection with the administration of KCNS.

There was also noticed in 9 cases (Series I, Series II, and Series III) a peculiar condition of the left ventricle, a blanching and hardening of a portion of the muscle of the left ventricular wall, this area lying approximately midway between the apex and the base of the ventricle and being of various sizes. When the heart was exposed before it ceased its contractions it was noted that such indurated areas failed to take part in the contractions. The presence of these areas of myocarditis did not seem to be concomitant with the appearance of vascular changes due to adrenalin, or if such changes were present, simultaneously, with the degree of these lesions. These lesions have been recently studied by Pearce.[7]

In regard to the histological changes we may, on the whole, refer to the description given in the paper by Loeb and Githens. The main additional facts are the following: (1) Injection of any of the chemical substances used in this work with adrenalin does not influence the histological character of the lesions produced. (2) Especially noteworthy in our specimens were a number of slides from an animal treated with iodipin and adrenalin showing the aorta broken, the break passing through the intima and media, and a polypoid fibrin thrombus attached to the site of this lesion; the blood had infiltrated the space between the media and adventitia and formed there the beginning of a false aneurysm. (3) In another animal treated with adrenalin, iodipin, and potassium iodide the blood had infiltrated the diseased media at some places, separated the elastic lamellæ and formed red-stained hyaline masses between the elastic fibers, the muscle nuclei disintegrating in this mass. (4) Van Gieson's stain, which was used among others in the preparation of these specimens, seems to have certain advantages over others. In good sections the periphery of the lesion in the media is clearly differentiated from the surrounding healthy parts by a red-stained area enclosing the necrotic yellow centre. (5) As Loeb and Githens stated in their paper, around the calcareous plates and places of necrosis we not rarely find a larger collection of nuclei than in other parts of the bloodvessels; this is perhaps due to the mechanical pressing together of the muscle nuclei about the site of the lesion. (6) In 1 case after iodipin and adrenalin injections, in which the aorta was normal to the naked eye, the aorta was likewise found to be normal under the microscope; it was, however, observed in this case, on microscopic examination that although the aorta was normal, one of the large vessels coming off from the arch of the aorta was markedly changed, showing even calcareous plates.

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[7] Experimental Myocarditis, Jour. Exp. Med., 1906, vol. viii.

In a microscopic examination of sections of the heart from areas showing the above-mentioned myocarditis, we note mainly degenerative myocarditic lesions.

As a main result of the histological examination of our specimens we regard the fact that neither iodipin, KI, nor KCNS produce the slightest variation from the usual lesions produced by adrenalin.

With the above results before us we are led to the following conclusions:

1. It is not possible, by the use of various iodine preparations, to prevent the arterial changes produced in rabbits by the injection of adrenalin. No beneficial effect of the iodine preparations was noted in our experiments.

2. When large doses of iodine preparations were injected the arterial changes produced by adrenalin were more marked than when adrenalin was used alone. Our experiments suggest that iodipin acts less strongly in this direction than does potassium iodide.

3. Injections of potassium sulphocyanide in either fair-sized or relatively large doses did not cause an increase in arterial lesions produced by adrenalin. Our experiments point rather to the possibility that potassium sulphocyanide exerts a preventive influence on the action of adrenalin in producing arterial lesions; we cannot state this fact with any degree of positiveness, for in view of the uncertainty and variability of the action of adrenalin our experiments are hardly sufficiently extended to draw a positive conclusion.

4. Iodipin has no advantage over bromipin in preventing the vascular lesions of adrenalin, and in none of these series (combination of adrenalin and iodipin or bromipin) do we find the lesions less marked than when adrenalin was used alone.

SO-CALLED HYSTERICAL AFFECTIONS OF THE ABDOMEN.

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I desire herewith to report two interesting cases, illustrating the diagnosis of certain affections presenting signs referable to the gastro-intestinal canal, and serving to illustrate the differential diagnosis between organic disease and gastro-intestinal hysteria. I am indebted to Dr. Stuart McGuire for the privilege of reporting the cases.

Case I.—Miss. W., aged nineteen years. Her father died of paralysis; her mother of cancer of the stomach; one aunt, aged thirty-eight years, died in May, 1905, after an operation for an abdominal tumor. There is no functional nervous disease in the family. The patient is the youngest of seven children, and aside from the ordinary mild infections of childhood was healthy until she was nine years old. She had pleurisy at the age of ten years, which confined her to bed four or five weeks; and whooping-cough four or five years ago. For the past five years she has had "dysentery" every summer, but was never confined to bed until 1905. (This was while she was affected with the present trouble.) For two or three years she thinks she has been the victim of ulcers of the bowel. She was never constipated until the onset of the present trouble. She uses coffee in moderation. For the past three years she has kept house steadily for her brother, but never did much work and never cared for "society." Menstruation appeared at eleven years of age. This function has been slightly irregular in time of appearance and the flow was often scanty or profuse from no apparent cause, but until the onset of the present trouble she never missed a period. For two years she has had occasional leucorrhœa. Almost ever since puberty she has been the victim of frequent "hysterical" paroxysms, characterized by screaming, crying, and apparent unconsciousness, sometimes lasting two or three days.

In January, 1903, while apparently in good health, she began to suffer severe, generally distributed abdominal pain, more marked in the right iliac fossa. This was colicky in character and associated with violent screaming and crying, but with no nausea or constipation, and no fever, as far as is known. For this she was blistered on her abdomen and back and kept in bed three weeks, during which time she improved; since then she has never been entirely well. In May, 1903, she had a second attack, from which she has suffered ever since, frequently having been confined to bed; within the past year has been a "semi-invalid." Throughout, her digestion has been in no way impaired and she has never vomited more than twice, and then from some such cause as improper diet or drugs.

In January, 1905, she was operated upon by a skilful surgeon for appendicitis, after which she remained in the hospital eight weeks (six weeks in bed). For two weeks more she was unable to walk on account of disability in the right lower extremity, and throughout convalescence her left arm was weak. Three weeks after going home (eleven weeks after the operation) the incision scar began to bleed (?) slightly and this kept up for a week. She then went to the country for two weeks to recuperate.

In May, 1905 (about this time her aunt died after an operation for abdominal tumor), she noticed for the first time that her abdomen was swollen. For this reason she consulted a physician who said he suspected "tumor and peritonitis," and referred her to another surgeon. The latter administered chloroform and told her upon recovery that the swelling entirely disappeared during anesthesia and that she had a "phantom tumor." She then returned to the country, where she remained six weeks. The swelling meanwhile persisted. Upon her return home she was attacked by "dysentery," which confined her to bed four weeks, during which time she suffered in addition to the abdominal swelling, abdominal pain, nausea, and vomiting. In August, 1905, she passed blood (?) from her bowels, on one occasion amounting to two tablespoonfuls (?), and once or twice after violent retching she noticed a little blood in the vomit.

On December 8, 1905, she became acutely constipated. For this she was confined to bed one week, and given purges, including jalap and croton oil, with little effect. After the administration of these she would be nauseated. Her bowels could be moved only by enemas. These were administered daily and followed each time by twenty-minute electric current to the abdomen. This treatment was kept up for nine days. Ever since this she has taken large enemas almost daily and often two or three times a day. The fecal material generally has been of liquid consistency (after enemas), though it is often well formed and in masses of normal size. A few times the bowels have moved without enemas. On the whole, since December, 1905, constipation has been obstinate, though there have been intervals during which it has been less marked, and for the past few days enemas are quite efficient. Defecation for months has been painful. Her appetite on the whole is good, but varies. Fruit acids are craved. Her usual weight is 135 to 140 pounds, and there has been no appreciable loss.

While convalescing from appendectomy she was unable to void urine for fourteen days, during which time she was systematically catheterized. In December, 1905, while constipated, she had to be catheterized twice, and again twice while she was being treated by electricity, but not since. The catheter was passed without difficulty.

There is generally severe pain, often lasting five minutes, at the end of urination. No hematuria has been noticed.

Since appendectomy was performed she has menstruated properly only three times, the last two times being in October, 1905, and January, 1906. At these periods the flow was free and apparently normal in duration. At other periods she has had only a very slight pink "stain." At each menstrual period, however, she has uterine colic and all the usual phenomena but the obvious flow. The hypogastric pain is more severe at each period than previously, and in February, 1906, confined her to bed one day. "Hysterical" paroxysms of crying and screaming have been frequent until the last two months, and for these morphine has occasionally been administered. She has been examined vaginally three times while conscious and this is neither pleasant nor painful. She has never had a sexual orgasm. She sleeps lightly and dreams much. On the whole she is miserable, but seems to have no apprehension of fatality of her affection. She is anxious to be cured, but does not desire to take chloroform, saying it makes her extremely nauseated. She says she abhors the idea of being operated upon.

Physical examination shows a well-developed, well-nourished young woman of good intelligence, refined and cultured manner, congenial and kind temperament. She is placid, gives fairly concise statements in her history, and is particularly exact in dates. She attributes most of her trouble to the operation, and her chief complaint is of obstinate constipation and abdominal swelling. The pupils are dilated, the face slightly flushed; she shows no evidence of pain, is comfortable in bed and perfectly able to walk. The plantar and patellar reflexes are exaggerated, but no pathological reflexes are elicited. There is a capillary pulse beneath the nails and on the forehead.

The abdomen is greatly distended, but the distraction is more marked below and above the umbilicus, with prominent hypogastrium, epigastrium, and flanks. At the level of the umbilicus there is a transverse constriction at the location of the lineæ transversæ. The circumference at the level of the iliac crests is 34½ inches, at the costal margin 27½ inches, and at the umbilicus 30¼ inches. Breathing is costo-abdominal in type and can be made wholly abdominal without undue effort or the slightest difficulty and does not produce pain. There is a small white scar over the appendicular region. The abdominal wall is held in extreme rigidity. In each iliac fossa and above the symphysis, tenderness is complained of upon deep palpation. In all parts of the abdomen rigidity can be overcome by prolonged broad pressure while she breathes deeply with her mouth open, shoulders elevated, and thighs flexed. The abdominal aorta, iliac arteries, and ascending and descending colon, can be palpated. No signs of free fluid can be elicited.

The distention gives the appearance of meteorism. This conclusion is confirmed by percussion, a tympanitic note being present all over the abdomen, save in the region of the liver. Hepatic dulness extends to the costal margin. Splenic dulness is not demonstrable. There is no abnormal area of circumscribed dulness, and no percussion tenderness. Auscultation reveals general exaggerated gurgling sounds due to peristaltic movements, though the latter are not visible. No fetal heart sounds nor movements are audible.

The rectal tube, finger, Kelley's large cystoscopic tube, and the proctoscope may be inserted into the rectum with difficulty. The latter meets marked resistance about 2 or 3 inches from the skin, but this is finally overcome and no further obstruction is noticed. The mucous membrane shows diffuse redness and obvious engorgement of vessels and is covered with mucous. There are many small erosions. The canal throughout contracts forcibly against the end of the instrument and the internal sphincter is greatly hypertrophied. One small, tender, and engorged hemorrhoid is seen at the mucocutaneous junction and the external sphincter is unusually tight.

Inspection of the external genitalia shows nothing abnormal, save slight leucorrhœa and hyperemia and a small urethral meatus. The clitoris is flaccid and normal in appearance. The hymen is present. Digital examination fails to detect any signs save great rigidity.

The heart, lungs, skin, lymphatics, bones, joints, and other structures are normal.

Immediately after the vulvar and rectal examinations the patient sunk into a stupor during which she was relaxed, breathed easily except for occasional spasmodic efforts and her pupils were dilated. There was no muscular twitching or crying. She responded to pain caused by flexion of the great toe and the passage of rectal instruments and was aroused without difficulty and made to walk around.

Diagnosis. Profound psychic hysteria; voluntary abdominal wall rigidity; general intestinal tympany; spasmodic stricture of the rectum; proctitis with erosions; inflamed mucocutaneous hemorrhoids; spasmodic amenorrhœa.

During chloroform anesthesia the abdominal distention entirely disappeared. She was allowed to regain consciousness and the distention reappeared.

Upon exploratory abdominal incision everything in the abdomen was found, upon careful examination, to be normal. During her stay in the hospital several weeks under active treatment for the psychosis, enemas had to be administered constantly, though occasionally a spontaneous bowel evacuation occurred. The distention persisted, though at intervals from no apparent cause, it would be slightly less marked. Since leaving the hospital she is apparently well.

This case affords a striking illustration of abdominal hysteria of profound type. The spasmodic stricture of the rectum may have been hysterical in origin or a reflex result of the painful hemorrhoids and proctitis.

Case II.—Miss Y. Her father, mother, two brothers, and one sister are living and well; there is some paralysis among her uncles, and her grandfather died of tuberculosis. She has had the ordinary infections of childhood, including diphtheria; pneumonia twice, the last time six years ago. About four years ago, at the time of her graduation from school, she had a mild attack of dysentery, confining her to bed a few days. She has always led an active life and was always remarkably healthy. She has taught school during the past four or five years.

In the latter part of the fall of 1902, while visiting away from home, she was seized one evening, while dancing, by an attack of severe colicky abdominal pain, nausea, and vomiting, so that she had to give up dancing. This pain was attributed at the time to dietetic error; the next day she was comfortable, save for general abdominal soreness. Within the course of a few days she was tolerably well, except for marked constipation. During the winter there were occasional recurrences of such paroxysms, and constipation became so marked as to necessitate purges.

Beginning March 17, 1903, she suffered for about a month with paroxysms of intermittent violent pain of the type of intestinal colic attended by nausea, obstinate vomiting, marked constipation, great abdominal distention, and with it all she was completely prostrated. She was operated upon at her home for intestinal obstruction. Upon opening the abdomen no obstruction was found, but the appendix, slightly adherent, was removed. During convalescence from the operation the distention and other symptoms persisted, and constipation was absolute for seven days. Upon getting up she noticed persistent abdominal distention. Her physician treated her almost continuously, especially for constipation, and was forced to administer enormous doses of strong purgatives. Licorice powder would generally be fairly effectual. The distention has persisted, the constipation has become more marked, and she has frequently suffered violent acute paroxysms of pain, nausea, vomiting, and prostration. In January, 1904, not having improved, she was operated upon again and her uterus, slightly retrodisplaced, was suspended.

She has continued to suffer recurrent paroxsyms of violent pain, vomiting, and prostration. Abdominal distention has persisted and she has not had a proper evacuation of the bowels in three years. She has had to continue taking purges and enemas, and came to St. Luke's Hospital for treatment.

Collateral Facts in the History. She has frequently noticed the passage of a little blood by the bowels, and on two or three occasions this amounted to a "couple of tablespoonfuls" of dark and clotted blood; she has noticed none of this during the past six months.

The evacuations have been made up largely of mucus, at times very marked quantities and in large flakes, especially after the second and third enemas. Purges produce violent increase of pain; enemas and the passage of rectal tubes are agonizing. There is never the slightest evacuation nor desire for such spontaneously; frequently two or three enemas are required and these are only partially successful. There has never been a formed movement. She has been treated constantly for hysteria, and for several weeks was cared for in a sanatorium in New York for this neurosis.

On one or two occasions she had pain of similar type, but having the location and radiation of right-sided renal colic. Her physician has found leukocytes, red cells, and small quantities of albumin in the urine. For the past year she has had dysmenorrhœa, and the administration of purgatives occasionally precipitates menstruation. She is otherwise well and hopeful. She has had no fever or chill, nor been unconscious. There have been no crying paroxysms, convulsions, or stupor.

Upon admission to the hospital she was suffering a violent attack, with great distention, rapid pulse, and other signs of a moderate degree of shock. After several days and repeated efforts a partial evacuation from the lower bowel was secured. Some time later, during the course of vaginal examination, the rectum was found impacted with feces. Examination of the pelvic organs was negative. During the first two or three days of June, 1906, she again suffered a violent attack similar in character to the above. Always after taking cathartic pills she suffered violently.

She is a well-nourished, slightly pale young woman, of a congenial temperament, but somewhat discouraged as to her recovery. She presents none of the appearances of a hysterical subject and seems perfectly normal in every respect except the abdomen. This is markedly distended all over and there is a transverse constriction at the waist line, that is, just above the umbilicus. Respiratory mobility is unimpaired. Measurements are as follows: At the xiphoid cartilage, 30 inches; half-way between the xiphoid and the umbilicus, 28¾ inches; at the umbilicus, 29¼ inches; half-way between the umbilicus and the pubis 32½ inches. There is some lordosis in the lumbar region, but this is due to prominence of the buttocks rather than to spinal curvature. There is slight general abdominal tenderness somewhat more marked on the right side. Nearly the whole of the colon is palpable, but none of the other abdominal organs can be felt. The abdominal rigidity is that only of distention. Percussion reveals a general tympany and diminished area of liver dulness; the splenic area cannot be outlined. There is no area of circumscribed dulness. Auscultatory percussion is entirely negative.

At this point a provisional diagnosis of incomplete intestinal obstruction was based on the following: (1) A history of recurrent attacks of violent abdominal pain attended with nausea, vomiting, moderate shock (prostration), and a number of times followed by the passage of blood; (2) obstinate, almost absolute, constipation; (3) intestinal distention; and (4) hypertrophy of the colon.

On rectal examination, externally no sign of the disease is seen; marked pulsation of the hemorrhoidal arteries is noted, and the rectum is empty. The passage of a proctoscope is attended by agonizing pain, in spite of the previous administration of a pint of olive oil. There is an area about 8 inches from the external sphincter in which there is greatly exaggerated tenderness and distinct resistance to the instrument, though this is finally overcome and the instrument passes 16 inches into the bowel. Inspection reveals an apparently sessile growth projecting into the lumen of the canal just above the junction of the sigmoid and the rectum. The mucous membrane of the rectum is moderately red, but shows no sign of localized disease and is empty. The sigmoid contains a small quantity of fecal matter and its mucous membrane is thrown into folds and apparently hypertrophied. There are no ulcers and only moderate inflammation. There are no signs of hemorrhoids, fistula, or fissure. The examination was agonizing to her, though she bore it bravely. The colic and local pain induced by the examination persisted until 4 P.M., at which time it was relieved by $\frac{1}{12}$ grain of morphine administered hypodermically.

Diagnosis. Incomplete intestinal obstruction; benign tumor of the colon?

Cœliotomy was performed June 11, 1906, by Dr. McGuire. The large intestine was distended with gas and feces; the rectum was empty. The sigmoid was found attached by a very short mesosigmoid causing sharp angulation of this tube. The colon above this point was filled with fecal matter and the rectum was empty. After dividing the short mesosigmoid the feces were easily manipulated into the rectum. Continuing the examination there were noted some adhesions of the omentum about the stomach. From the sense of touch it was impossible to find any lesion of the mucous membrane.

What we believed to be a growth arising from the mucous membrane as seen through the sigmoidoscope proved to be an invaginated portion of the mucous membrane of the sigmoid flexure through the portion pulled upon by its short mesentery and causing angulation of this part of the gut. The uterus was held anteriorly by an artificial ligament about an inch long resulting from a previous ventrosuspension. The old scar was dissected and the abdominal wall united in layers.

Convalescence was uninterrupted and on the third day following the operation a painless bowel evacuation was secured by the administration of 2 drams of extract of cascara followed by a simple enema. At the present time she is entirely free from symptoms. This woman had been treated three years for hysteria.

REVIEWS.

Pediatrics. The Hygienic and Medical Treatment of Children. By Thomas Morgan Rotch, M.D., Professor of Pediatrics, Harvard University. Fifth edition. Pp. 1060. Illustrated by numerous engravings in the text and by colored plates. Philadelphia and London: J. B. Lippincott Co., 1907.

The necessity for a new edition of Dr. Rotch's well-known textbook, according to the preface, "has afforded an opportunity to introduce many changes designed to bring the subject-matter up to the standard required by recent advances in the investigation of disease and the modern methods of treatment." The work upon the third edition, however, had been so thoroughly done that Dr. Rotch has really found little to change.

The division on feeding naturally has received the greatest attention, and the most recent advances in our knowledge of the chemistry of milk and the physiology of digestion have been incorporated in it. It is interesting to note that Dr. Rotch's attitude toward home modification has become distinctly less hostile than of yore, even though his championship of laboratory feeding is, and rightly, quite as enthusiastic. Top-milk feeding receives scant courtesy and Chapin's useful little dipper is not even mentioned. The subject of whey modifications has been considerably elaborated and their use is advocated as preferable to cereal attenuants.

One of the most important changes is the adoption of an etiological basis of classifying diseases of the gastro-enteric tract, those of the stomach and of the intestines being separately divided as they are developmental, non-infectious, or infectious, with intestinal worms as an independent class. This seems to us a distinct advance over previous systems of classification and worthy of general adoption for its simplicity and scientific accuracy.

A few changes have been made in the terminology of diseases of the kidneys, as the substitution of "acute degeneration of the kidney" for the older and less accurate term, "acute hyperemia."

Some necessary additions have been made in the section on diseases of the blood, and the term "articular rheumatism" is banished in favor of "rheumatic fever," as laying too much emphasis upon the articular manifestations of what is actually a general infection. New sections upon atrophic and hypertrophic diseases of the joints and upon infectious arthritis, with a short section on the hypertrophic pulmonary osteo-arthropathy of Marie, about complete the changes to be found after a very careful comparison of the old and new editions. We may also call attention to the emendation which has been consistently made throughout the revision by the substitution of the plural of the first personal pronoun for the singular, an alteration presumably dictated only by the author's modesty, but which destroys to a considerable degree the inspiration of the personal note that has been such an attractive feature in Dr. Rotch's previous writings.

T. S. W.

A Manual of Obstetrics. By A. F. A. King, M.D., Professor of Obstetrics and Diseases of Women in the Medical Department of the George Washington University, Washington, D. C., and in the Medical Department of the University of Vermont. Tenth edition. Pp. 688, 30 illustrations, and three colored plates. Philadelphia and New York: Lea Brothers & Co., 1907.

When, as in the case of King's *Manual of Obstetrics*, the publishers announce that it begins its second quarter-century with its tenth edition, what more need a reviewer say? The hypercritical are disarmed by the passage of time; commendation seems almost superfluous, since few books, and no other obstetrical book extant, can claim such a record. Dr. King's abilities and long service as a teacher have brought him into intimate personal relations with a large proportion of the practitioners of this country; indeed, he is now teaching the sons of some of his earlier students. To others he has become well and favorably known by his book—which in its tenth edition must claim an even wider circle of friends. The general scope of the work remains, as from the first, elementary, the main object being such brevity and simplicity of statement as might be easily intelligible to all students. In the new edition the chapter on fecundation and nutrition of the embryo has been almost entirely rewritten; extensive changes have been made in the chapters on pelvic deformities, cutting operations upon the mother, mutilating operations upon the child, placenta prævia, and puerperal septicemia; and minor alterations have been made elsewhere throughout the book. It is a pleasure to commend a work which, in addition to the authority and dignity of age, exhibits also the freshness of rejuvenescence, and to hope that Dr. King in the future, as in the past, may continue to issue new editions of his really excellent *Manual*.

A. K.

Progressive Medicine. A Quarterly Digest of Advances, Discoveries, and Improvements in the Medical and Surgical Sciences. Edited by Hobart Amory Hare, M.D., Professor of Therapeutics and Materia Medica, Jefferson Medical College; assisted by H. R. M. Landis, M.D., Demonstrator of Clinical Medicine, Jefferson Medical College, Philadelphia. Vol. I, March, 1907. Pp. 280. Philadelphia and New York: Lea Brothers & Co., 1907.

This, the first volume of the series for 1907, opens with a splendid chapter, one hundred and twenty-one pages, on recent progress in the surgery of the head, neck, and thorax, by Charles H. Frazier. Of the many important subjects discussed the following seem especially worthy of mention: Intracranial injuries, which includes an excellent discussion of 530 fractures of the base of the skull; Crile's method of controlling hemorrhage during operations on the head; the operability of cerebral tumors, including the technique of different operations; the treatment of cerebral abscess and of tumors of the hypophysis and the cerebellum; the surgical treatment of epilepsy and trifacial neuralgia; the treatment of carcinoma and syphilis of the mouth and tongue; the technique of operations on the neck; diseases of the thyroid and the parathyroid, including a discussion of the results of Kocher's latest 1000 thyroidectomies; carcinoma and other tumors of the breast, including a new method of removing the breast; the diagnosis of infections of the mediastinum; the surgery of the lung; and the results of sixteen recent cases of suture of the heart. Robert B. Preble discusses recent progress in infectious diseases, devoting special attention to the role of insects in the transmission of different infectious diseases; the importance of blood cultures in the diagnosis of certain infections; a report of 561 cases of diphtheria; the antitoxic treatment of dysentery; an excellent discussion of some recent contributions to epidemic cerebrospinal meningitis; pneumonia; rheumatic fever; scarlet fever; tuberculosis; typhoid fever, etc. Floyd M. Crandall, discussing diseases of children, devotes special attention to the incidence of disease in children—a review of 1000 cases; infantile mortality; the urine of infants; the tonsils as portals of microbic invasion; hemorrhages in the newborn; nervousness in children; infant feeding; the management of infants during hot weather, etc. D. Braden Kyle discusses recent progress in rhinology and laryngology, devoting special attention to deflections of the nasal septum; the treatment of hay fever; disease of the maxillary sinus; syphilis of the nose and the upper air passages; nasal and pharyngeal growths; the treatment of pharyngitis; the bacteriology of a common cold; tuberculosis, etc. B. Alexander Randall, reviewing recent literature on otology, dwells especially upon diseases of the labyrinth; meningitis; brain abscess; phlebothrombosis; middle-ear suppuration; mastoid diseases, etc. Throughout the work seems to have been done with care and discrimination; assuredly the volume will well repay careful reading.

A. K.

THE IMMEDIATE CARE OF THE INJURED. By ALBERT S. MORROW, A.B., M.D., Attending Surgeon to the Workhouse Hospital and to the New York City Home for the Aged and Infirm. Pp. 340; 238 illustrations. Philadelphia and London: W. B. Saunders Co., 1906.

THERE is always a place for a comprehensively written and well-illustrated book on the immediate care of the injured which can be put into the hands of those members of the laity who are likely to be called upon to administer the first treatment in the case of injuries, such as nurses, policemen, steamship officers, soldiers, superintendents of factories, etc. The great difficulty in this matter is to teach such persons not to be overconfident or attempt to do too much. Surgery is a subject in which a little knowledge is sometimes a very bad thing, especially when it is put into practice. The reader of the present volume should carefully consider the preface, in which it is distinctly shown that non-professional aid should be only temporary, and nowise supersede or take the place of proper medical or surgical attention. Another feature which should be carefully considered by the layman who undertakes to administer first aid is the questions of anatomy and physiology. One-third of the present volume is devoted to this subject, and it is presented in simple and comprehensive language. Considerable space is devoted to bandaging, and also to accidents and emergencies.

We have but one criticism to make, and that applies to the treatment of wounds. We do not feel that the author has laid sufficient stress upon the prime importance of surgically clean hands. This is the most frequent source of infection of all wounds, and the layman especially needs to have the fact impressed upon him. The method of cleansing the hands is mentioned in detail, but its importance is not dwelt upon sufficiently.

In every respect this book can be recommended as a reliable guide not only to the layman, but also to nurses and to recent graduates of medicine. J. H. G.

OUTLINES OF HUMAN EMBRYOLOGY. By GEORGE REESE SATTERLEE, M.A., M.D., Instructor in Histology and Embryology in the University and Bellevue Hospital Medical College, New York. Pp. 173; 99 illustrations. New York: John Wiley & Sons, 1906.

THIS little book, designed by the author for use in the laboratory and as a means of rapid review, is one of a class begotten by the modern methods of overcrowding the medical student. To fulfil the demand for condensed or boiled-down information the book, without doubt, is of value, but in default of a series of detailed lectures or demonstrations with the microscope it would be wellnigh unintelligible. This is especially true of the chapters covering the earliest development of the embryo, where comparative embryology is introduced to such an extent as to make the vaguest possible impression as to what really probably does occur in the human animal. Of course, it should be remembered that it is extremely difficult to create a conception of this portion of the subject in the mind of the student without full detailed descriptions, and in this case these have been sacrificed to brevity. The later chapters, in which the development of the various organs is discussed separately, are handled more clearly than the earlier ones. The author's style is clear but overly concise, and it may be said that throughout there is a marked tendency to sacrifice all else to brevity and condensation. There are numerous evidences of hurried proof-reading, and some inaccuracies in measurement. The illustrations are clear, well reproduced, and in the main well chosen, but it is questionable whether the rather numerous photomicrographs are of much value to the class of readers for which the volume is designed.

H. R. A.

PSYCHOLOGY APPLIED TO MEDICINE. INTRODUCTORY STUDIES. By DAVID W. WELLS, M.D. Philadelphia: F. A. Davis Company, 1907.

THIS small volume of 130 pages is an attempt, as stated in the preface, "to bridge over the gap between psychology and medicine." The title would suggest a thorough correlation of the two subjects. In the bridging process, however, both subjects are connected in a superficial and unscientific manner. That branch of medicine to which psychology has been most extensively applied is treated with a lack of thoroughness and knowledge that is little short of amazing. As an example of this, on page 121 the author states that "neurasthenias are difficult cases to treat, as they are hard to hypnotize. The same applies to hysteria." It is, of course, well known that hysterical subjects are always selected for medical experiments on account of the ease with which they yield to hypnotic suggestion. In another paragraph there is a quotation from a newspaper report of an interview with a New York doctor concerning "his reviving of a moribund patient who had called for him previously to sinking into the comatose state." The author's knowledge appears to be limited to his own specialty, ophthalmology, from which most of his illustrations and comparisons are drawn. The other branches of medicine are either entirely neglected or simply mentioned as quotations from articles by others.

A reviewer should be charitable with any book which attempts to widen the range of therapeutic measures. The application of psychology to the treatment of disease has been too much neglected in the past. There is, indeed, a crying need for just such a book as the title would indicate. This volume, however, does not only not fill this need, but is likely to have a pernicious influence on account of its lack of thoroughness and accurate information.

D. J. McC.

A Pocket Formulary. By E. Quin Thornton, M.D., Assistant Professor of Materia Medica in the Jefferson Medical College, Philadelphia. Eighth edition. Pp. 287. Philadelphia and New York: Lea Brothers and Co., 1907.

The new edition of Thornton's *Formulary* has been improved in many particulars, and it has been revised throughout to conform to the new edition of the U. S. Pharmacopœia. This is of major importance, since in the new Pharmacopœia the strength of many potent remedies has been materially changed. In the formulary diseases are arranged alphabetically, facilitating ready reference. Special mention should be made of the indications for use appended to many of the formulæ, and the suggestions to a choice when several formulæ are given under the one heading. Due regard has also been given to the palatability and the compatibility of the different formulæ. The little book has subserved a useful purpose in the past; in its new and revised edition it should commend itself to a still wider circle of practitioners. A. K.

A Text-book of Obstetrics. By Barton Cooke Hirst, M.D., Professor of Obstetrics in the University of Pennsylvania. Fifth edition. Pp. 915; 753 illustrations, 39 of them in colors. Philadelphia and London: W. B. Saunders Co., 1906.

Dr. Hirst's *Text-book of Obstetrics* has become so widely known and so highly prized that mere mention of a new edition suffices.

The many excellencies of the book are common knowledge, so that a detailed recital thereof is quite superfluous. In this the fifth edition many minor modifications have been made, but especial attention has been paid to recent advances in our knowledge of puerperal infection and gestational toxemia. Dr. Hirst's large and continuously broadening experience as an obstetrician fits him eminently to prepare a book upon the subject which in this new edition must enlist the interest of an increasing number of practitioners. A. K.

ATLAS OF CUTANEOUS MORBID HISTOLOGY. By MAX JOSEPH, M.D., Physician for Skin Diseases in Berlin, and J. B. VAN DEVENTER, M.D., Oberstabsarzt of the Netherlands East Indian Army in Batavia-Java. 53 colored figures. Chicago: W. T. Keener & Co., 1906.

TIME was, and that not so very long ago, when the student of cutaneous diseases had to depend upon the meager paragraphs to be found in text-books of dermatology and of general pathology for information concerning the morbid anatomy of the skin. But *nous avons changé tout cela;* cutaneous pathology is no longer an unexplored region and text-books and atlases dealing with this special field increase yearly, thanks to a host of earnest workers whose labors are not only illuminating the dark places in the pathology of the skin, but are teaching the general pathologist some things worth knowing.

In this new *Atlas of Cutaneous Morbid Histology* there are 53 figures in colors, representing 45 separate diseases, which are, for the most part, accurate illustrations of disease of the skin as seen under the microscope. In the text which accompanies the plates a brief account of the most important histological features, which characterize the diseases portrayed, is given. We notice that the authors have given a new name, hydrocystoma tuberosum multiplex, to the disease first described by Kaposi as lymphangioma tuberosum multiplex. We cannot feel as certain as the authors seem to be that this neoplasm is of sweat-gland origin.

The chief, indeed the only, adverse criticism we have to make of this atlas is that too many figures (more than a fourth) are devoted to comparatively uncommon affections, such as acanthosis nigricans, pemphigus foliaceus, psorospermosis follicularis vegetans (which, by the way, might have been much better named), ichthyosis hystrix, etc., while so common a disease as eczema is not represented at all, the figure bearing this title being, in fact, a variety of ringworm. This necessarily limits the usefulness of the atlas very much.

M. B. H.

PRINCIPLES AND APPLICATION OF LOCAL TREATMENT IN DISEASES OF THE SKIN. By L. DUNCAN BULKLEY, A.M., M.D., Physician to the New York Skin and Cancer Hospital. Pp. 130. New York: Rebman & Co., 1907.

THE contents of this volume comprise a short series of lectures delivered to physicians at the New York Skin and Cancer Hospital. The book does not pretend to be a treatise on the local treatment of skin diseases, but it gives practical suggestions that are not ordinarily met with in text-books. The author, as is well known to the profession, has had an immense experience with these diseases, and therefore all that he says is of value. While the local treatment is especially death with, attention is everywhere directed to the importance of the internal treatment suitable to the case.

One cannot peruse the volume without learning much of the principles of treatment, and after all is said concerning drugs, combinations, and formulæ, in stubborn, deeply rooted inflammatory diseases of the skin, success will not crown the efforts of the physician if the principles of practice be neglected or ignored. For permanent relief the patient and not the disease is to be treated. The booklet is a sort of *vade mecum,* from which many practical points may be gleaned. It is indexed in such a manner that everything referred to can be readily found. L. A. D.

A TEXT-BOOK OF OPHTHALMIC OPERATIONS. By HAROLD GRIMSDALE, M.B., F.R.C.S., Ophthalmic Surgeon and Lecturer on Ophthalmic Surgery to St. George's Hospital; Surgeon to the Royal Westminster Ophthalmic Hospital, London, and ELMORE BREWERTON, F.R.C.S., Ophthalmic Surgeon to the Metropolitan Hospital; Surgeon to the Royal Westminster Ophthalmic Hospital, London. Chicago: W. T. Keener & Co., 1907.

THE various operations upon the different ocular structures are clearly described in this work, simple diagrams rendering the descriptions easily intelligible. A feature of the book is the number of operations proposed by recent writers, which are fully described, with the authors' names. Thus twenty-three operations for cicatricial entropion are mentioned, with the names of their proposers, and most of these are described with sufficient accuracy.

The work for exhaustiveness and wealth of detail, of course, is hardly to be compared with Czermak's upon the same subject, but it will prove a reliable guide for ophthalmologists who desire a work in English dealing with operations upon the eye.

T. B. S.

PROGRESS
OF
MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF
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REGIUS PROFESSOR OF MEDICINE, OXFORD UNIVERSITY, ENGLAND,
AND
W. S. THAYER, M.D.,
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The Difference in the Physical Signs at the Pulmonary Apices.—SEUFFERHELD (*Beit. z. Klin. d. Tuberkulose*, 1907, vii, 65). Considering the importance which is being attached to even slight changes in the physical signs at the apices as an indication of beginning pulmonary tuberculosis, it is necessary to have well in mind the normal differences. In the examination of 120 apparently healthy individuals between the ages of sixteen and fifty, Seufferheld found in 90, or 75 per cent., a lower position of the right apex; in 71 cases, the difference between the two sides was 1 cm. or less; in 19 cases over 1 cm. In 30 cases the apices rose to the same level, but in 8 of these 30 cases the diagnosis of a lesion at one apex could not be definitely excluded. The lower position of the right apex was associated with a slightly tympanitic quality to the percussion note, and usually a little impairment. Expiration at the right apex was accentuated, prolonged, and in some instances approaching the tubular quality, in 97 cases, or 80.8 per cent.; equal on the two sides in 18, or 15 per cent. In 5 cases it was rougher and more prolonged on the left side, but all of these 5 belong to the group of 8 suspected cases. Increased bronchophony was noted on the right side in 26 cases, or 21.7 per cent.; on the left, in 2, or 1.7 per cent.; and muscle sounds, that is, sounds which were inconstant and could not be satisfactorily explained otherwise, in 12 cases, or 10 per cent. The breath sounds were different on the two sides in 7 of the 30 cases of equal position of the apices. In all other instances the difference in expiration followed the percussion differences. In those cases in which the variations were so marked as to arouse the suspicion of a pulmonary lesion, radioscopy and the orthodiagraph were resorted to. Of 33 cases examined, 30 showed an equal position of the two apices and 28 an equal transparency. The 5 cases showing differences again belong to the group of 8 suspected cases. On inspection the right shoulder was noticed to droop a little when viewed from the front in a large percentage of instances, but as a rule there was no difference when viewed from behind. In these cases the clavicle ran more horizontally than on the left side, and by measuring the sternoclavicular angle Seufferheld has sought to get an index of these differences. Of 90 cases measured the left was larger than the right 53 times; the right larger than the left, 20 times; equal, 17 times. Anatomically, and as shown by the orthodiagraph, the pulmonary apices have an equal position. Seufferheld suggests three factors which may be of importance in changing the percussion note so as to simulate a lower position on the right side: (1) Scoliosis which is frequently noticed in the cervical region associated with drooping of the right shoulder; (2) the greater muscular development on the right side; and (3) the compression which occurs between the resistance offered by the liver to expansion and the rigid shoulder girdle. The tympanitic quality which so frequently accompanies the percussion note and the changes in the character of the breath sounds are referred to the arrangement of the bronchial tree, the higher point of departure of the right main bronchus and its shorter course before division and the higher point of departure of the eparterial bronchus and its closer relation to the apex.

The Dicrotic Pulse in Aortic Insufficiency.—JANOWSKI (*Ztschr. f. klin. Med.*, 1907, lxi, 121); NURNBERG (*ibid.*, 1907, lxi, 130).—It is characteristic of the typical collapsing pulse in aortic insufficiency that there is no recurrent wave on the catacrotic limb. The question arises if the diagnosis of pure aortic insufficiency would be compatible with the presence of a dicrotic wave. As is well known, dicrotism depends upon decreased tension of the arterial wall, and it will occur in the pulse if the conditions exist to produce a diminished tension. Such conditions are the acute infectious diseases, dyscompensation of the cardiac activity, vasomotor disease as in morbus Basedowii, and in certain special poisonings, as by amyl nitrite. When such conditions occur in aortic insufficiency, the pulse becomes dicrotic and this need not disprove the diagnosis. Goldscheider has recently published 3 cases of aortic insufficiency with dicrotic pulse and a systolic mitral murmur, 2 of the 3 cases showing at autopsy a combined aortic and mitral insufficiency. On the basis of thess cases and of the observation previously published by Geigel, Goldscheider considers the mitral insufficiency responsible for the dicrotism. Geigel's observation was on a young woman with aortic insufficiency who, during an attack of acute endocarditis associated with the development of mitral insufficiency, acquired a dicrotic pulse which became again typically quick, as during convalescence the signs of mitral insufficiency receded. Janowski points out that in all of these cases there was fever, and in order to decide which of the two factors is the more prominent each must be studied separately. He gives a large number of instances of combined aortic and mitral lesions with careful pulse tracings, and in none does the mitral insufficiency influence in the least the typical collapsing quality. In fever the pulse of pure aortic insufficiency frequently becomes dicrotic and Janowski believes this to be the cause of the dicrotism in the cases of Geigel and Goldscheider. He takes exception to Goldscheider's view that when the pulse of aortic insufficiency becomes dicrotic we can diagnosticate the establishment of mitral insufficiency. Nürnberg, on the basis of his observations, supports the view of Goldscheider. He considers four factors of importance in causing the dicrotic notch: The presence of mitral insufficiency, the presence of fever, the age of the patient, and the duration of the lesion, the two last factors being an index of the elasticity of the arterial wall.

The Nature of Diabetes Insipidus.—Seiler (*Ztschr. f. klin. Med.*, 1907, lxi, 1) communicates a fairly complete and extensive article dealing with the nature of diabetes insipidus. The work in many ways is a confirmation of the results of work of E. Meyer (*Deut. Arch. f. klin. Med.*, 1905, lxxxiii). Seiler's work, however, was done in the years 1903–04; so about simultaneously with the work of Meyer, and each is, therefore, confirmation of the other's results. Seiler's observations were made on 4 cases. The first one was a girl, fourteen years of age, who had suffered from a fracture of the skull, following which there was bitemporal hemianopsia and diabetes insipidus. It is probable that the injury to the hypophysis played no role, as the blood pressure was not increased. The second case was one of diabetes insipidus in a child of eight years, in which no etiological factor could be determined. The third case was in an eighteen-year-old man, in association with fracture of the base of the skull. The fourth case was in an hysterical girl sixteen years old. This was the only case in which the course was favorable, and in which marked improvement was observed.

The old view that diabetes insipidus is a disease in which there is increased excretion of urine without any disease of the kidney can no longer be held, and for some time many persons at least have believed that the condition is due to a disturbance of the kidney function. This view is based mainly on the experiments of Strubell, Tallqvist, Meyer, and others. It was to confirm this view that the observations of Seiler were undertaken.

The first observations consisted in the accurate administration of constant quantities of water at hourly intervals during the twenty-four hours of the day, with examination of each separate specimen of urine voided. It was found that the amount of urine passed at night is considerably larger than that passed by day, but that the specific gravity of all specimens is quite constant. Such a condition can only result from an anomaly of kidney function, and is entirely opposed to the view of a disturbance of absorption, since it is hard to see why absorption should occur more rapidly by night than by day. Next, comparative estimations were made of the urine in a case of diabetes insipidus as compared with that of a normal person, and the differences in excretion were observed after the administration of a large amount of water, $1\frac{1}{2}$ liters, early in the morning. Results showed that there was much more prompt water excretion in the healthy child than in the one with diabetes insipidus. In the patient with diabetes insipidus the excretion of the excessive amount of water extended over a longer period; in the healthy child the increased excretion was very quickly manifested, within a few hours. This, too, speaks for a disturbance of kidney function, unless it depends upon a disturbance in water absorption. To see whether it is due to a disturbance of absorption, accurate estimations of the hemoglobin, by means of the Fleischl-Miescher hemometer, were made before and after drinking the large amount of water. It was found that in the patient with diabetes insipidus, just as in the healthy person, there was quite a marked diminution in the amount of hemoglobin in the blood, and therefore a thinning of the blood, appearing twenty minutes after the drinking of the water. From this it is quite evident that the delayed water excretion in the diabetic patient, as well as the increased amount of night urine as compared with day urine, cannot be due to delayed absorption, but must depend upon disturbance of kidney function.

A second series of observations was made to determine the nature more accurately of the disturbance in kidney function which occurs in diabetes insipidus. First, the quantitative estimation of the uric acid excreted; second, of urea; third, of posphates, and fourth, of chlorides, was made. Under ordinary circumstances there was no variation in what is considered the normal twenty-four-hour amount, except in the case of uric acid, in which there was a definite and quite marked diminution.

Further observations were made to determine the rapidity of excretion of certain urinary constituents. For this purpose, at a certain time during the day, large amounts of one of the various constituents of the food was administered and the effect of this upon the various urinary constituents was determined. In these experiments careful controls were made with the normal. In the first experiments the effect of the administration of large amounts of albumen was tested. The patient received in the morning five eggs, and afterward hourly observations of the urine were made. Now, while in the healthy the taking of five eggs was followed in from four to six hours by a marked increase in the specific gravity of the urine, which must depend upon an increased urea excretion, such a result did not occur in the case of diabetes insipidus, but there the increased excretion of urea occurred gradually, without any real change in the concentration of the urine. Next, an increase of carbohydrates in the shape of levulose and dextrose was given, without, however, there occurring any abnormal behavior of kidney activity under these conditions. The excretion of large amounts of salt was studied by the administration of large amounts of potassium iodide and the observation of the rate of its excretion in the urine. With the patient taking the ordinary amount of water, no apparent difference in excretion, as compared with that in the normal person, occurred. While Seiler lays no stress upon the excretion of methylene blue as an indication of normal or abnormal kidney function, he nevertheless made observations in order to render his experiments complete, and showed that methylene blue was excreted in the same way in the diabetes insipidus case as in the normal, except for a slight lessening in the time of excretion.

Now a series of more exact metabolism experiments was undertaken, in which the nourishment remained constant and the amount of water administered was varied. With the patient taking all the water he desired, the metabolism protocol showed no marked variation from the normal, except the diminution in uric acid previously mentioned. The amount of water was then limited, and a control experiment was made on a healthy child. In the healthy child it was found, in confirmation of the observations of Neumann, that with an increase or diminution in the amount of water administered, nitrogen loss or retention occurs for the first one or two days, and that after that the figures are about those as before the change was made. In the diabetes insipidus case, however, following the restriction of water, the results were different. The amount of nitrogen excreted remained permanently diminished, and the same was true of chlorides and phosphates. The amount of uric acid excreted was in all conditions about the same, constantly under the normal. Also the excretion of potassium iodide under these conditions of water restriction was delayed, and, while with active diuresis the total amount of potassium iodide excreted through the kidneys was 65 per cent., with restriction of the water it was only 28 per cent. With the restriction of water, also, the patient experienced certain symptoms, such as headache, lassitude, disturbance of appetite, and tendency to vomiting, symptoms which were similar to those of beginning uremia. With a diminution in the amount of fluids given, there also occurred a definite increase in the osmotic pressure of the blood serum.

All these observations point quite definitely to diabetes insipidus being due to a disturbance of kidney function; that is, that the kidneys are no longer in a position to excrete a urine above a certain concentration, which concentration is usually considerably less than the normal. A satisfactory excretion of the organic and inorganic constituents of the urine then only takes place when such an amount of water is given that the urine will be of low concentration. If this amount of water is not given, the retention of the solid constituents occurs, and symptoms appear which are similar to those of uremia. The body, therefore, requires an increased amount of water, which the patient recognizes by increased thirst, and so arises the main symptom of the disease—polydypsia. Seiler draws attention to the possibility that the polyuria associated with contracted kidney may in its ultimate nature be the same as that of diabetes insipidus; that in both cases the polyuria is a compensatory mechanism, enabling the body to get rid of the solid waste products satisfactorily. In one case the disturbance of kidney function is due to an interstitial nephritis, and in the other it depends upon nervous conditions, at present unknown.

The therapeutic indications in this condition, therefore, are not the limitation of the amount of water administered as is so frequently recommended, but an attempt to stimulate the kidney to excrete the solid constituents in a more concentrated condition. Theoretically, therefore, the use of diuretics might be indicated. Experiments were made with the administration of caffeine, agurin, and theocine, but with none of these, however, were satisfactory results obtained. It was impossible by these means to obtain any greater or more active excretion of the solid constituents. With theocin the excretion of urea was both absolutely and relatively diminished. It is, therefore, evident that this drug might do more harm than good. Seiler lays stress upon the necessity in the indiviudal case of finding the optimum amount of fluid to be administered, and warns against the danger of a large increase or diminution above or below this amount.

A New Test for Sugar.—Fenton (*Lancet*, 1907, i, 215) outlines a new test for sugar which may be applicable in urinary examinations in certain cases, especially as it enables one to distinguish the hexoses from the pentoses or other lower sugars. The test is of great delicacy, but requires some little skill in manipulating. By it as low as 0.2 per cent. or less of sugar in the urine can be detected. The reaction depends upon the fact that all carbohydrates of the hexose or polyhexose type (such as dextrose, levulose, cane sugar, milk sugar, or maltose) yield a certain amount of bromomethyl-furfural, CH_2BrC_4, H_2O-CHO, when acted upon by hydrobromic acid under appropiate conditions; and, further, that the latter substance reacts with malonic ester in presence of alkalies, giving rise to a product the solutions of which exhibit a powerful blue fluorescence. The test is as follows: Pour 4 to 5 c.c. of urine on an excess of solid anhydrous calcium chloride so as to form a semisolid or pasty mass. Add 10 c.c. of toluene containing 2 to 3 drops of phosphorus tribromide, and boil for a few minutes (bearing in mind the inflammable nature of the toluene). Pour off the toluene solution and, after cooling, add 1 c.c. of malonic ester and a little alcohol. On neutralizing with alcoholic potash a characteristic pink color occurs. The mixture then should be diluted considerably with alcohol and a few drops of water, when it will give a beautiful blue fluorescence if sugar be present.

The Pathogenesis of Congenital Icterus in the Adult.—Chauffard (*La semaine médicale*, 1907, No. 3, 25) reports the case of a young man, twenty-four years of age, a brunette, and of a somewhat delicate build, whose skin and conjunctivæ were jaundiced. There was no family history of jaundice or of liver trouble. When very young he had diphtheria and at twelve years of age was extremely weak and anemic. This was followed a year later by an increase in the jaundice. At eighteen he had abundant and repeated epistaxis during the warm weather. The jaundice had been noted by the father of the boy after birth and had persisted steadily, but at times was more or less pronounced, being striking after fatigue or while travelling. The effect of any diet on the jaundice was not noticed. He did not take alcohol. As a rule, the patient was not sick, but at times after fatigue he had severe, cramp-like pains in the abdomen and increase in the jaundice. The stools were never clay-colored and the urine had showed only urobilin. There was no pruritus, no cutaneous hemorrhages or hemophilia. The reflexes were normal, pulse normal. Other organs were negative. The liver was not enlarged, but the spleen was quite large and hard. This enlargement was noted at the age of nine years, never disappeared, and remained the same up to the present time. Chauffard then gives a *resume* of the literature on congenital icterus, grouping them under three classes, in two of which the liver or changes in the bile-ducts are responsible for the jaundice, and a third group, to which the present case belongs, in which there were no changes in the liver. The history of this latter group starts with an article by Minkowski, "A Peculiar Hereditary Form of Splenomegaly with Chronic Jaundice, Urobilinuria, and Renal Siderosis" (*Verhandl. d. XVIII Kong. f. inn. Med.*, Wiesbaden, 1900). The most probable explanation, according to him, is that there is some anomaly in the destruction of the blood, influenced by a primary lesion of the spleen. Other cases resembling this group are reported by Bettman; Gilbert, Castaigne and Lereboullet; Widal and Ravaut, Pick, and von Krannhals. (See AMER. JOUR. MED. SCI., 1905, cxxx, 721.) Chauffard then discusses two possible causes of this condition—(1) a persistent angiocolitis or (2) splenohemolysis—the first of which he dismisses on account of the clinical history of the case, its prolonged character, the few or only vague symptoms of an infection, the action of fatigue on the aggravation of the symptoms, and the condition of the liver in a case that came to autopsy, indicating little or no change in the biliary tract. He takes up the indications of hemolytic action in the reported cases and then passes to an extensive study of the blood in his own case, especially on the corpuscular resistance to hemolysis, following the work of P. Ribierre (*Thèse de Paris*, 1903) by the use of solutions of sodium chloride in different dilutions, and finds the hemolysis increased by comparison with other cases of simple jaundice and in those in which the liver is involved and not the spleen. Chauffard concludes that the predominating role in this condition is played by the spleen, and suggests that this study may lead to the recognition of the importance of splenohemolysis and the etiology of chronic jaundice in the adult.

Influenza.—The whole of volume lxxviii, No. 1, 1907, of the *Practitioner* is devoted to influenza, and contains seventeen articles by some of the celebrated English writers. DONALD ARMOUR, in his article on influenza and appendicitis, quotes extensively from the literature on this question, the most convincing statistics being those of Gagnière, who found that in 900 cases of influenza the percentage of appendicitis was about 1.3, these cases appearing during and ceasing, apparently, with the epidemic. BOTTOMLY notes an apparent increase in the number of cases of appendicitis during years when influenza was prevalent. The mortality from appendicitis appears to be increased during these years. According to other statistics this relation varies somewhat during different epidemics, being more prevalent when the influenza epidemic has many gastro-intestinal features. JOHN COWEN, in writing on the cardiac complications of influenza, notes that the myocardium is most commonly involved. The myocardial weakness is manifested by its usual train of symptoms, and the danger of sudden overstrain should be especially borne in mind. The treatment and prevention are then considered.

WILFRED HARRIS, in writing on the nervous system in influenza, divides the symptoms into: (1) Those at the onset, such as sudden faintness or syncope, headaches, severe general pains, sweats, and chills. (2) Those occurring in the course of the illness, meningitis, ocular disturbances, hysteroid convulsions, myelitis, Landry's paralysis, and neuritis. (3) Among the sequels, loss of the power of smell and taste, progressive bulbar paralysis, myasthenia gravis, epilepsy, loss of memory, and inertia of mind and body. T. CLAY SHAW, in discussing the psychoses of influenza, says the prognosis is good. "As a rule, the subjects, whether melancholic, maniacal, or stuporous, eventually and quickly recover, but a certain number do not and these go on to dementia or remain in a more or less excited chronic condition. Upon all who are concerned with the treatment of influenza the obligation rests to remember that in proportion as the patient is neurotic or degenerate, or has a bad family mental history, so he is more likely to suffer from a postinfluenzal psychosis, which in a small percentage of cases may end fatally, and which is always a matter of solicitude, because of the tendency to relapse and of the frequency of suicidal influence."

Calcium Salts in Pneumonia and Heart Disease.—SIR LAUDER BRUNTON (*Brit. Med. Jour.*, 1907, i, 616) contributes a short article on the use of calcium salts as cardiac tonics in pneumonia and heart disease. He refers to the discovery by Ringer of the beneficial effects of calcium salts on the heart. Brunton has had occasion to use calcium chloride in several cases of pneumonia with weak hearts, with apparently some beneficial results, and wishes it to have a wider trial. He prescribes it in 5 to 10 grain doses every four hours, and disguises the bitter taste by minute doses ($\frac{1}{20}$ grain) of saccharin to about 10 grains of the calcium salt. Brunton has used it also in cardiac cases with apparently good results. The chloride acts more quickly, but the lactophosphate or glycerophosphate could also be used. He thinks the good effects of a milk diet in some cases with heart disease may be due to the calcium salts contained in the milk.

Diagnostic Doses of Tuberculin.—ROEPKE (*Ztschr. f. Tuberkulose*, 1907, x, 412). The method of giving tuberculin for diagnosis according to the directions of Koch, namely, 1 mg., 5 mg., 10 mg., and again 10 mg. at intervals of two days, has been recently modified by employing much smaller doses. Loewenstein suggested giving $\frac{1}{5}$ mg. and repeating that amount four times, and Moeller, Ostrowsky, and other eminent authorities, after extensive trial, have approved the method. Roepke contends that such small doses are entirely insufficient and cites a number of instances in which no reaction has followed as many as five injections of $\frac{1}{5}$ mg. and appears in a perfectly typical manner when larger doses are resorted to. Some of these cases were known to have pulmonary tuberculosis; others were early, doubtful cases. He considers the method time-consuming and very unreliable, and returns with some modifications to the older procedure. The principal point of discussion is the maximum dose to which we must proceed. Koch's directions call for a repetition of the 10 mg. dose, and Bandelier insists upon following the directions faithfully. Other authors, as Junker, suggest stopping at 5 mg.; the latter's scale is $\frac{1}{10}$, $\frac{1}{2}$, and then 5 mg. Roepke, in a long discussion with many statistics, accepts a routine similar to this, giving $\frac{1}{5}$, 1, and then 5 mg. This gives results he believes as reliable as any other method, and shortens the time necessary for the test. One point of especial interest is the statement that he obtains signs of a local reaction in about 40 per cent. of the cases. This is an exceptionally large percentage.

SURGERY.

UNDER THE CHARGE OF
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The Technique of Narcosis.—STRAUCH (*Zentralbl. f. Chir.*, 1907, xxiv, 233) says that the use of chloroform is gradually becoming more restricted in favor of ether. He has employed morphine and alcohol as preliminaries to the anesthetization, as advised by Wenzel, but now prefers another method. Nothing affects more injuriously the heart strength than the continuous loss of sleep, anxiety, and excitement preceding operation, and in a large number of cases Strauch has found, on the morning of operation, arrhythmia of the pulse. That such a heart can offer no great resistance to the veil effects of chloroform is clear. To obviate this difficulty he gives to the patient, the evening before operation 1 gram of veronal. Even the most excitable women sleep, without exception, usually to be waked about an hour before operation, when they receive a hypodermic injection of morphine and a dose of alcohol. The veronal frequently continues to act, so that under its influence, with that of the morphine and alcohol, the patient is sleeping when brought into the operating-room, and is anesthetized quietly with a small quantity of ether given by the drop method. When necessary a few drops of chloroform are administered.

The Treatment of Pancreatic Fistulæ.—HEINEKE (*Zentralbl. f. Chir.*, 1907, xxxiv, 265) says that the two conditions that make these fistulæ so troublesome are the macerating effect on the skin of the pancreatic secretion and their persistency. To control the first, at the Leipzig Clinic, the siphon apparatus of Perthes for empyemas has been employed with excellent effect. The surrounding skin remains intact and the healing of the fistula hastened considerably. Wohlgemuth has shown that the quantity of pancreatic secretion produced depends upon the composition of the food taken. A pure fat diet produces the least, an albuminous diet a greater quantity, and a carbohydrate diet considerably increases it further. Acids increase, while alkalies decrease it. Wohlgemuth cured such a fistula in a short time by placing the patient on a diabetic diet and giving small doses of sodium bicarbonate. Heineke reports a case in which a similar result was obtained.

Subperiosteal Resection of the Diaphysis in the Long Bones.—SMOLER (*Ztschr. f. Heilkunde*, 1907, xxviii, 1) describes his method of operating as follows: The diseased diaphysis is exposed by a longitudinal incision so placed as to do as little damage as possible to the muscles, vessels, and nerves. The periosteum is then carefully separated from the bone and the bone is resected by a chisel or a Gigli saw, the epiphysis being avoided in young people. It has been his practise to pack the wound with gauze and to apply to the extremity a wooden or a metal splint. In limbs with one bone, by this method, it was difficult to preserve the correct line in the reformed bone, and thus to obtain a good functional and cosmetic effect. The employment of weight and extension for the same purpose is associated with difficulty in the change of the dressings, and causes the patient much pain. Smoler accomplishes the purpose by the use of an aluminum splint introduced into the wound, to bind together, to a certain extent, the two ends of the bone during the process of union. The presence of this foreign body, by its irritation, aids the periosteum in forming new bone. He does not employ absorbable material for the splint, and does not entirely close the wound, because sooner or later these are generally extruded. He has had no cases in which an insufficient regeneration of new bone occurred, as shown by the *x*-rays and the functional results. By the use of only a few sutures and large drainage tubes the discharge finds a free escape, and the wound may be washed out by a hydrogen peroxide solution. When sufficient regeneration of new bone has taken place the aluminum plate is sawed or broken with forceps into several fragments and is removed piecemeal through the drainage opening. A half-year or more was usually required for healing.

The Treatment of Undescended Testicle.—LOTHEISSEN (*Ztschr. f. Heilkunde*, 1907, xxviii, 37) gives the following reasons for operating for undescended testicle: Pain and neuroses are frequent. Periorchitis occurs easily (from frequent trauma) and can prove dangerous. Hernia is frequently associated and may become incarcerated. Torsion of the cord may take place and lead to gangrene of the testicle, unless operated on promptly. Psychical disturbances are frequent on account of the "lack" of the testicle. Operation may permit the further development of the testicle, so that the earlier the operation is done the better. After it is done the patient may enter the army. The cosmetic effect deserves some attention. If the testicle is healthy, orchidopexia should be done, while castration should be reserved for a necrosed testicle or malignant degeneration of the testicle.

Lotheissen makes an incision as for an inguinal hernia, two fingers' breadth above Poupart's ligament, but extends it past the spine of the pubis to about the root of the penis. He then opens the inguinal canal as for a hernia operation, being careful not to injure the testicle or its bloodvessels. The hernial sac or tunica vaginalis is exposed and isolated from the surrounding structures as far as the internal ring, where it is ligated. It is then divided and removed from the testicle. The spermatic cord is then stretched carefully. Lotheissen has been able thus far to do this without excision of the vessels. The testicle is then placed in a cavity in the scrotum made with the finger, and is held there by a gauze tampon. The internal oblique and transversalis muscles, sometimes the rectus also, are sutured to the lower internal edge of Poupart's ligament, over the cord. The gauze tampon is then removed from the scrotum and the connective tissue is sutured over the cord, so that the whole cord is enclosed in a canal. It will be observed that as the sutures are being introduced and tied the testicle is being forced lower until it reaches the lowest part of the scrotum. In bad cases it may remain a finger's breadth higher than on the opposite side. The external oblique fascia and the skin are then sutured. Care should be taken to avoid, as far as possible, infection by the urine.

Tumors of the Kidney in Children.—MOUCHET (*Annales des maladies des organes génito-urinaires*, 1907, xxv, 342) says that they are observed especially in the first years. Some of the tumors are of complex structure, formed essentially of embryonal elements, multiple tissues in a sarcomatous network. Some are true cancers, in the clinical sense of the word, and are rapid and progressive in development. The tumor element is the first and often the only clinical sign. Hematuria is rare and tardy. The volume reached is generally considerable. Death occurs, usually from cachexia, in a short time. Extirpation is difficult and grave. An early diagnosis and immediate operation are essential to the radical cure of a malignant tumor. The first is difficult because of the absence of the important symptom of hematuria. In operating the abdominal route should be preferred, but it is essential to have good light and to remove the tumor *en masse*, that is, the tumor, its fatty capsule and the glands, thus avoiding transplanting the tumor debris in the wound. The prognosis, however, for a permanent cure is so bad that one can say of them that the chief interest in these tumors concerns their anatomical structure.

Some Notes Apropos of Cases of Appendicitis Treated during the Years 1905 and 1906, in the Service of J. Verhoogen, in the Hospital Saint Jean.—GRAEUWE (*Jour. de chir. et Annales de la Société Belge de chirurgie* 1907, vii, 140) says that 36 cases were treated in this time, with 2 deaths, or a mortality of 5.5 per cent. The 1 case of simple acute appendicitis was treated only medically and recovered. This consists of rest, an icebag locally, and no opium, except a hypodermic injection of morphine at the beginning of the attack, if the pain is acute. After some weeks without fever, and when there is no tenderness on pressure and the swelling has disappeared, the operation is usually done. An analysis of the blood is always made to determine the presence of an active suppurating focus. In some cases, as when the patient supports a family and the necessity for going back to work is urgent, he sometimes intervenes in a shorter time, but even in these only after the fever has disappeared some days. In all cases from the time of their entrance into the hospital, numerous leukocyte counts are made. It is especially in the cases with localized peritonitis that these counts are valuable. In these there is a high leukocytosis during the first days, which increases as the abscess progresses and decreases as the abscess diminishes. If, in an acute case, there is marked rigidity and a daily increase in leukocytosis, operation is done immediately. The abscess is opened and drained with rubber tubes and gauze. Irrigation of the cavity of the abscess is not practised for fear of infecting the general peritoneal cavity. The search for the appendix is made with care. If it can be liberated without too great separation of adhesions it is removed, if not it is left in place.

If, on the other hand, the leukocytes decrease and the general condition remains good, operation is delayed. Thanks to the examinations of the blood he has never had cause to repent for having operated too hastily or too tardily. In cases of generalized peritonitis, with anxious expression, frequent vomiting, and irregular pulse and temperature, operation is done immediately. After resection of the gangrenous appendix a free discharge of the pus is provided for by several incisions, which are left largely open and through which are introduced large drains surrounded by iodoform gauze.

The Pathology of the Effusion in Traumatic Serositis.—MERCADE and LEMAIRE (*Revue de chirurgie*, xxvii, 396) find that the lymphatic elements are the dominating factor in the effusion, the blood elements being of so negligible a quantity that we may infer that in some cases it may be wholly lacking, only the lymph remaining. They conclude, therefore, that the traumatic effusion is purely of lymphatic origin, aside from that blood which comes from rupture of the capillaries, and which at the end of some days is well mixed with the lymph elements.

Pylorectomy in Benign Stenoses of the Pylorus.—BRECHOT (*Revue de chirurgie*, 1907, xxvii, 471) says that the gravity of a pylorectomy varies with that of the lesion of the pylorus for which it is done. It is followed by immediate results, as good as those of gastro-enterostomy, in those cases in which the resection is easy. The later results are not as troublesome as those of gastro-enterostomy, nor do the complications arise, often fatal from the evolution of the causal lesion. When a pylorectomy is impracticable, a gastroduodenostomy should be done, since it gives a more physiological result than a gastro-enterostomy. The latter will be necessary when the two previous operations cannot be done. The duodenum is an essential organ in digestion. It permits the passage only of the necessary quantity of chyme. It controls the intestinal and pancreatic secretions. Brechot gives a series of cases, personal and from other writers, to show—that the evolution of the ulcer is not arrested by gastro-enterostomy; the dangers of the operation; the influence of a secondary resection; and the necessity of resecting the ulcer, and reëstablishing the gastro-intestinal canal.

The Perineal and Freyer's Transvesical Prostatectomies.—CASTANO (*Annales des maladies des organes génito-urinaires*, 1907, xxv, 401) says that the suprapubic is more easily executed than the perineal prostatectomy. It has the great advantage of exposing the bladder to direct examination by sight, and of facilitating the extraction of vesical lobes and calculi, so frequent in these cases. In total prostatectomy, which should always be done, the perineal operation sacrifices the posterior urethra, while it is left intact in the transvesical. Catheterism is very easy after the transvesical; after the perineal sounds must be passed for some time to shape the urethra. The duration of the transvesical operation is shorter and the rectum is in less danger. Impotence and epididymitis are rare in the transvesical and common in the perineal operation. The great advantage of the latter is the better drainage afforded, and when it is impossible to disinfect the bladder Castano considers this operation preferable. The functional results are more rapid after the transvesical operation, urination being soon spontaneous and complete. Aside from the severely infected cases, Castano believes that the results obtained from the Freyer operation are on a par with and probably better than from the perineal.

The Incision for Appendectomy and Lateral Celiotomy.—BARACZ (*Zentralbl. f. Chir.*, 1907, xxxiv, 330) says that partly as a result of his visit to America, in 1902, and partly as a result of his own experience, he considers the McBurney incision preferable to all others in preventing ventral hernias. He is convinced that it is of great service in other operations in the lateral abdominal regions, as for the making of an artificial anus in the cecum or sigmoid flexure, a better sphincter being obtained than by the other incisions.

The After-treatment of Operations for Cancer of the Breast.—EWALD (*Zentralbl. f. Chir.*, 1907, xxxiv, 385) suggests a method of after-treatment in these cases which aims to reduce limitation of movements in the shoulder following these operations. The affected arm is not bound to the side of the body in the usual manner, but is held in abduction beyond a right angle, by means of a traction apparatus. This can be made by adhesive plaster wrapped about the wrist and hand, which grasps a suitable piece of wood from the two ends of which a cord or bandage attaches the hand to an apparatus fixed above the head of the bed and projecting toward the affected side of the body. An additional support is applied under the arm just above the elbow. The patient is kept in this position for three or four days, and after that the arm is gradually lowered until when the patient is ready to leave her bed it is at the side. Whenever she goes back to bed the arm should be again raised in the abducted position. By this method Ewald found that except in excessively obese individuals, the patient, two weeks after operation, could place her hand on her head by her own strength. The stretching of the skin over the axillary space, in this position, prevents the collection of blood or lymph in the cavity made by cleaning out the axilla.

Spontaneous Abdominal Herniæ through the Semilunar Line of Spiegel.—THEVENOT and GABAURD (*Rev. de chir.*, 1907, xxvii, 586) say that the diagnosis concerns itself particularly with the volume of the hernia. If it is small the patient complains only of the functional troubles, as dyspepsia, enterocolitis etc. On account of the fixed pain associated with it, one thinks on the left side of a sigmoiditis, and on the right side of a cholecystitis or a chronic appendicitis, according to the height of the hernia. The hard mass in the abdominal wall makes one think of a fibroma, lipoma, or abscess. The diagnosis, usually, will be made only by the reducibility, impulse on coughing, etc. If the hernia is large, the diagnosis is usually easy, and it should then not be confounded with an inguinal or umbilical hernia. The treatment is essentially the same as that of an umbilical hernia.

An Anatomicopathological Study of Dislocations of the Semilunar Cartilages of the Knee.—DAMBRIN (*Rev. de chir.*, 1907, xxvii, 616) says that in recurrent dislocations operation is necessary, as well as in recent dislocations that cannot be reduced by the usual manipulations. The operation is at the same time benign and efficacious. It consists either of an arthrotomy with fixation of the cartilage, meniscopexy, or extirpation of the cartilage, meniscectomy. The incision is a vertical one, about 6 cm. long, and about midway between the patella and the internal lateral ligament, with its centre over the interarticular line. This avoids wounding the important internal lateral ligament. The cartilage is then sutured to the periosteum of the tibia or is removed. In comparing the results of the two operations Dambrin says that both are equally easy, and while both are very benign extirpation is more so than fixation. Of 87 extirpations there were 4 in which very mild infection occurred, while of 35 cases of fixation there were 5 in which joint infection occurred, 1 of which was prolonged. This difference is accounted for by the prolonged manipulations in placing the sutures in fixation and the presence of the sutures as foreign bodies. In 53 of the 87 extirpations absolute cures were obtained. Lenail found in dogs that the meniscus was regenerated after its removal. Dambrin does not believe that this occurs. From the point of view of efficaciousness, the results of extirpation of the cartilage are easily superior to those of fixation.

Transportation of the Sartorius Muscle as a Means of Fixation in Excision of the Knee.—Kofmann (*Zentralbl. f. Chir.*, 1907, xxxiv, 417) says that posterior flexion of the knee as a late result of excision is a well-known condition. None of the many methods of excision which have been devised have succeeded in overcoming this difficulty. Heusner recommended the transference of the flexors of the knee to the anterior surface of the knee. Kofmann tried this several times and felt that he had done too much. He concluded that in his next case he would merely transplant the sartorius as Schantz had done for dislocation of the semilunar cartilage. In this case he made a median incision through which he exposed and prepared the sartorius from the middle of the thigh to its insertion into the anterior surface of the tibia. He then made a transverse incision from the end of the first incision over the tuberosity of the tibia to the lateral ligament, thus opening the joint. This was the seat of a partly fibrous and partly bony ankylosis. The patella and the ends of the femur and tibia were removed to permit the placing of the limb in extension, and the sawed surfaces were kept approximated by silk sutures through the periosteum and cartilage. The sartorius was then attacked. It was easily seen that it was so loose that it was unnecessary to isolate the muscle, but that a little loosening was sufficient to permit displacing it from its normal lateral position to one directly over the front of the joint. Three sutures secured it over the rectus muscle. Kofmann satisfied himself of the effective strength of the displaced muscle by lifting the extremity by the heel, the bones remaining in position without any support. The skin sutures were introduced, and the dressing and a plaster cast were applied. They were removed in eight days; healing was by first intention; a lighter cast permitted the patient to go around. In fifteen days all dressings were removed.

THERAPEUTICS.

UNDER THE CHARGE OF
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The X-rays in Diseases of the Blood and Blood-forming Organs.—Pancoast (*Univ. Penna. Med. Bull.*, 1906, vol. xix, No. 11) reports his own observations upon this subject and gives a review of the literature. In regard to leukemia he finds that of 63 patients reported, only 4 (6.35 per cent.) are still alive and well three to six years after the primary symptomatic cure. He assumes that it is safe to assert that, were the final reports at hand of all patients that have been treated by the *x*-rays, this percentage would be but slightly altered. In 39 patients, or over 70 per cent. of those that died or are at present in critical condition, there was either a very marked amelioration in the symptoms or a symptomatic cure, as a result of the treatment at first. In view of these results, Pancoast considers that the Röntgen ray can hardly be held to be a specific in leukemia and that the effects of treatment by it are not very encouraging. He asserts that the dangers attending the use of the rays should be remembered and evidences of toxemia should be looked for; frequent urinary examinations should be made to determine the extent and rapidity of tissue destruction and the state of the kidneys. There is clear evidence that death has been caused or hastened by failure to observe these points. In favor of the treatment of pseudoleukemia by means of the *x*-rays Pancoast mentions the following points: (1) A cure for at least three or four years may be obtained in about 25 per cent. of the patients treated. (2) In almost all instances not so cured, life is prolonged during a period of comparative comfort by improvement in the general condition or by the relief of pressure. (3) No other agent can yield as good results. (4) By further improvements in technique and a better understanding of the pathology of the disease, even more favorable results than those obtained at present may be possible. In the treatment of polycythemia by the *x*-rays the results cannot be considered very favorable. In the absence of more definite knowledge it would seem that the rays should be applied over the splenic area. Twelve instances of splenic anemia have been collected by Pancoast in which improvement due to the employment of the Röntgen rays has been noted. With regard to pernicious anemia the statement is made that before subjecting the patient to the rays, even for the purpose of skiagraphy, except in urgent necessity, metabolism investigations should be carried out in conjunction, and the physician must be sure that there are no evidences of toxemia and that other forms of treatment are unavailing. Unless these precautions are taken the results may be of the most untoward character.

The Roentgen Rays in Mediastinal Tumor.—ELISCHER and ENGEL (*Deut. med. Woch.*, 1906, No. 40, p. 1620) report 3 instances of mediastinal lymphoma treated with the x-rays. They conclude that if the tumor is directly retrosternal it is most amenable to this mode of treatment. In the first patient after eight treatments of from five to ten minutes' duration there was evident improvement; after sixteen sittings the patient, considering his cure complete, ceased treatment. One year later there were slight signs of recurrence, but the patient refused further treatment In the second instance the lesion seemed to be a tuberculous adenitis. The exposures brought about marked relief of the symptoms, but the tumor persisted A similar result was attained in the third patient, the nature of whose tumor was undiagnosticated. Four other patients are reported in whom the treatment was ineffectual; the tumor in these instances was possibly a lymphosarcoma. The authors consider that all mediastinal neoplasms should be submitted to radiotherapy; in many instances a rapid betterment will follow, and, if the tumor is simple lymphoma, a cure may be, perhaps, expected.

Quinine in Influenza.—BROADBENT (*The Practitioner*, 1907, lxxviii, 13) has held, since the first invasion of influenza, that quinine is the best remedy. He prefers to give it in a prescription containing 1 part of ammoniated quinine to 2 parts of liquor ammoniæ acetatis. Of this mixture the dose is 3 drams every hour for three hours and then every four hours. In the fulminating attacks of the disease, in which the patient has become comatose, quinine hydrobromide given hypodermically in large doses has relieved the unconsciousness. As a prophylactic the author orders 2 grains of quinine every morning during the prevalence of epidemics and considers that he gets good results; although patients who were taking quinine occasionally contract influenza, in many instances this makes a complete difference in the patient's liability to infection.

The Treatment of Serous Pleurisy.—FORCHHEIMER. (*Jour. Amer. Med. Assoc.*, 1907, xlviii, 28) considers that the longer fluid is allowed to remain in the pleural cavity the greater the likelihood of the formation of adhesions, that it is a risk for the patient to go about his ordinary pursuits with the effusion, for dangerous symptoms may develop at any time, and finally if the fluid is withdrawn the process may be terminated. These Forchheimer considers strong arguments for early paracentesis, and he would not wait for the occurrence of the symptoms of intrathoracic pressure. He also advocates withdrawal of the fluid in tuberculous pleurisy since, after tapping, an artificial hyperemia, following the pressure anemia previously existing, occurs and good effects, as claimed for it by Bier, take place. The hyperemia also induces a leukocytosis, which is followed by the development of connective tissue which tends to encapsulate the tubercle. He makes the definite statement that paracentesis should be performed eight days after compression of the lung has existed, and as much fluid as possible should be drawn off, for the more fluid withdrawn the greater the hyperemia.

The Treatment of Hay Fever.—BELBEZE (*La province médicale*, 1906, No. 34, p. 404) gives the following list of the drugs useful in this affection: Cocaine, stovaine, or adrenalin with cocaine, applied to the inferior turbinates; cologne-water or lavender-water, either combined with menthol or pure, by inhalations; stramonium by inhalation or in cigarettes; vaseline with menthol, applied directly or as a spray; ammonium chloride as an inhalation. Cocaine and stovaine should be employed with caution on account of the possibility of habit formation. In the prophylactic treatment of the condition the author advocates the use of sodium cacodylate for about a month previous to the time when an attack is expected. Intranasal conditions should receive appropriate treatment, and potassium iodide is also useful as a preventive when given in doses of about 8 grains daily for two or three weeks before an anticipated seizure.

The Treatment of Eclampsia.—LAURANDEAU (*Jour. de med. de Paris*, 1906, No. 38, 412) advises in addition to the usual means—chloral, chloroform, the bromides, etc.—doses of 15 drops of fluid extract of veratrum, which may be accompanied by hypodermic injections of $\frac{1}{60}$ grain each of scopolamine hydrobromate and morphine hydrochloride, given deep into the muscles of the thigh and repeated in thirty minutes if necessary. Two hours later, if the symptoms persist, a third injection and a second dose of veratrum may be given. Laurandeau advises against exceeding these doses. The alkaloid veratrine may be substituted. The inunction into the abdomen of an ointment consisting of veratrine $7\frac{1}{2}$ grains, cocaine hydrochloride 6 grains, vaseline one ounce, is also suggested.

PEDIATRICS.

UNDER THE CHARGE OF

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Alcoholic Cirrhosis of the Liver in Children.—ERNEST JONES (*Brit. Jour. of Children's Diseases*, January, 1907, p. 1) prefaces a critical study under this caption by reporting two personal cases of unusual interest. The first was that of a Russian Jewess, sixteen months old. She was brought to the hospital for manifestations of malnutrition of three or four months' standing, and was gradually getting worse. The mother had been in a hospital in St. Petersburg for several months and the child had been cared for by its grandmother from the age of eleven months. During the whole of this time the child had received a little wine twice a day; the amount of the dose varied, but was frequently a tablespoonful. Wasting, vomiting, looseness of bowels, and slight jaundice were the prominent symptoms. There was a little fluid in the peritoneal cavity; the liver was enlarged, reaching to within an inch of the umbilicus, its edge sharp and hard. The spleen was just palpable. The course of the disease after treatment was begun was very favorable, and recovery took place. The second case was that of an English girl, three years old. She had had pertussis, which had begun six months previously and had lasted three months. Toward the end of that time brandy had been prescribed in doses of a dram twice a day. This amount was exceeded to a variable extent and was continued for over five weeks. There was considerable fluid in the peritoneal cavity, and the liver extended three or four fingers' breadth below the costal margin; the spleen was not palpable. The subsequent course of this case was very favorable. Jones has collected 72 other cases from the literature as a basis of a very careful analysis of some of the points concerning this condition. As to frequency of incidence the available statistics show that the greater frequency of the condition in adults is by no means so pronounced as is generally supposed. The commonest source of the alcohol—in over half the cases—was the parents, who usually gave it with the good intention of improving the child's condition—for debility or minor ailments. In the rest of the cases the commonest source was the medical practitioner, and in 3 of the cases the medical attendant specifically ordered an increase of the habitual alcoholic beverage with the idea of treating the malnutrition due to the unrecognized cirrhosis. In conclusion Jones expresses the opinion that the evidence as to the importance of alcohol in the causation of hepatic cirrhosis in children is sufficient to justify our taking the possibility of its production into consideration when ordering alcohol or permitting its employment in the case of children who are amenable to the influence of our advice.

Hot Intestinal Injections in Enterocolitis.—Caravassili, of Athens (*La Clin. Infant.*, August, 1906, p. 475), advocates the use of hot intestinal injections in enterocolitis as being more efficacious than simply warm injections or other local measures. The temperature should be from 104° to 107.6° F. Colic and spasm are diminished and sometimes entirely suspended for from four to nine hours after such an injection, and then reappeared generally in much less severe degree. The character of the stools is promptly improved and their frequency diminished after each injection, and usually the number required does not exceed from three to six for any given case.

Statistics of Diphtheria from the Paris Hopital des Enfants-malades.—Since assuming direction of the diphtheria pavilion of this hospital in 1901, Marfan (*Revue mensuelle des maladies de l'enfance*, April, 1907, p. 180) has had published each year, by one of his internes, the statistics of his service. These reports, which have been made successively by Weill-Hallé (*Soc. méd. des hôp.*, June 12, 1903), Leenhardt (*ibid.*, January 22, 1904), Detot (*ibid.*, December 2, 1904), Le Play (*ibid.*, December 8, 1905), and Henri Lemaire (*ibid.*, February 15, 1907), are of extreme value to anyone making research of the literature of diphtheria, and illustrate strikingly the efficacy of the antidiphtheritic serum. Previous to 1894, when the serum was first employed, the mortality from diphtheria in all the Paris hospitals for children averaged

50 per cent. As compared with this the following statistics for the years from 1901, in Marfan's service, are peculiarly significant. By reduced mortality is to be understood that calculated after the exclusion of cases dying in less than twenty-four hours after admission. For 1901-02, 1122 cases, total mortality 21 per cent., reduced mortality 12.3 per cent.; for 1902-03, 1048 cases, total mortality 15.8 per cent., reduced mortality 11.1 per cent.; for 1903-04, 605 cases, 14.2 and 9 per cent.; for 1904-05, 561 cases, 7.6 and 5.47 per cent.; and for 1905-06, 534 cases, 10.11 and 6.97 per cent.

In explanation of the higher figures of the first two years it is stated that toward the end of 1900 a very severe epidemic of malignant diphtheria prevailed and that the usual number of cases treated was more than doubled, and that this epidemic lasted until near the end of 1902. From these statistics Marfan believes the mortality from diphtheria depends particularly upon the frequency of malignant anginas. As accessory factors in the mortality must be considered the number of croups necessitating intervention, the frequency of bronchopneumonia, and that of certain associations, as shown in the slight increase of the mortality observed in 1905-06, depending upon the frequency of association with measles. It is to be understood that the accuracy of these observations presupposes the methodical employment of the antitoxin, the lack of which, Marfan believes, stands as the principal factor in the mortality of the disease.

Purulent Meningitis and Empyema of the Frontal Sinuses in the Course of Eruptive Fevers.—SCHOLLE (*Arch. f. Kinderheilk.*, 1906, xliv, 306) reports two observations, one of measles and scarlatina in a boy of ten years and one of scarlatina in a girl of six years, in both of which death ensued from purulent meningitis consecutive to empyema of the frontal sinus, which was discovered only at the autopsy. In both cases the inflammation of the frontal sinus had been so intense as to cause obstruction of the nasofrontal canal and to prevent the outflow of pus by the nose, which would have pointed to the correct diagnosis. In the first case the pus accumulated in the right frontal sinus was under great pressure, since the child presented during life an œdema of the right eyelid and exophthalmos, symptoms which where wrongly attributed to a thrombosis of the venous sinuses consecutive to a suppurative otitis which existed at the same time. As diagnostic signs Scholle suggests tenderness on pressure on the floor of the sinus, occasionally with a sensation of yielding of the wall and a flow of pus into the nose; and redness after percussion over the sinus, more marked and persistent than over the sound side.

The Duration of Immunity after Injections of Antitoxin.—SITTLER (*Jahrb. f. Kinder.*, 1906, xiv, p. 442) bases this study upon 912 cases at the Children's Hospital at Strassburg immunized by an injection of 500 units; 700 of these were healthly children, the brothers or sisters of 378 diphtheria patients; 212 were scarlatina patients, who were in a different ward from the diphtheria cases. Of the 700 cases 4 contracted diphtheria sixteen hours, thirteen days, five weeks, and six weeks respectively after injection. Of the scarlatinal cases only 1 developed a mild diphtheria three days after injection. Sittler comes to the following conclusions: (1) The prophylactic injection of serum produces an immunity of from three to five weeks or even longer only under the condition that the child is not brought in contact too often with diphtheria cases or convalescents from the disease. In case of such contact, when the child is constantly exposed, the duration of immunity is not greater than from ten to fifteen days. But under these same conditions non-immunized children contract the disease very much more frequently than those who have been immunized. (2) All catarrhal affections and others which affect the mucous membranes, even in immunized cases, constitute a condition eminently favorable to the development of diphtheria, and shorten at the same time the duration of immunity. (3) After diphtheria occurring in children who have been previously immunized, a second attack of the disease may occur as early as with children who have only submitted to immunization. (4) Generalized scarlatiniform eruptions, even when they develop without fever and without marked angina, are most frequently true scarlatinal infections.

Serum Treatment of Diphtheritic Paralysis.—COMBY (*Arch. de méd. des enf.*, August, 1906) reports further cases illustrating the value of antidiphtheritic serum in the treatment of diphtheritic paralyses. He advocates the free use of the serum, whether or not it had been previously used in the treatment of the primary disease. The dose should be large and repeated if necessary.

The Blood Changes in Mumps.—Referring to the difficulty in diagnosis encountered at times at the outset of a case of mumps, IRA S. WILE (*Arch. of Pediat.*, September, 1906, p. 669) states that a blood examination will positively determine the diagnosis in its earliest stage without waiting to observe its course. As a result of examinations in 20 consecutive cases he concludes that lymphocytosis, both relative and absolute, is noted on the first day of the disease, no matter how small the tumor may be, and continues until all swelling has disappeared. It is more marked relatively with bilateral involvement than with unilateral disease, and is most marked in children at puberty, though it is present in adult cases, but is less accentuated. Eosinophiles are slightly decreased in the beginning, but rise to normal or above during convalescence. The eosinophilia is higher in bilateral involvement than in the unilateral affection, as the period of convalescence begins at this time. The basophiles are not affected. The polynuclear neutrophiles vary relatively inversely with the lymphocytes. With orchitic complications the polynuclear neutrophiles tend to increase relatively, though there may be no absolute leukocytosis. Hyperleukocytosis does occur, but is not of high degree. There is no secondary anemia. Lymphocytosis is a diagnostic feature of mumps, differentiating it from adenitis. The average count of this series was: Lymphocytes, 59.37 per cent.; polynuclear neutrophiles, 38.32 per cent.; polynuclear eosinophiles, 2.28 per cent.; polynuclear basophiles, 0.43 per cent. It is important to remember that at one year the lymphocytes normally form 54 per cent. of the leukocytes; at three years, 43 per cent.; at six years, 38 per cent.; at eight years, 35 per cent.; and at nine years and over, 31 per cent. As cases of mumps in children under one year are very rare, it is a safe general rule to state that a relative lymphocytosis occurring in children to the extent of 50 per cent. or above is always pathological. The total leukocyte count may be said to vary normally from 10,000 to 15,000 in the first year and a half of life. After the second year the number approximates that found in adult life—8000. In adenitis, even from decayed teeth, there is a high leukocytosis in children—even as high as 43,000.

OBSTETRICS.

UNDER THE CHARGE OF

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The Induction of Labor for Disproportion between Mother and Child.—Moller (*Arch. f. Gynäk.*, 1906, Band lxxx, Heft 3) reports 80 cases of induced labor for disproportion between mother and child. The total number of births among which these cases occurred was 21,066; among these there were 646 contracted pelves, or 1 in 32.6, which is about 3 per cent. In all labors occurring in the clinic, induction was practised once in 277.2 cases; among the contracted pelves it was necessary to induce labor once in 8.73 cases. The most frequent type of contracted pelvis was the justominor. Labor was induced in 2 cases for contraction of the birth canal outside of the pelvis. In these cases the head presented in 67.5 per cent.; in 32.5 per cent. the breech presented. Among the cases of head presentation, 14 had unfavorable position of the cranium, and 10 of these were cases in which elastic bags had been used. Of these induced labors 52.5 per cent. terminated spontaneously, and 47.5 per cent. required an obstetric operation to complete delivery. The maternal mortality among these patients was 1.25 per cent. The maternal morbidity was 33.75 per cent., or 27 cases. Among these were 12 cases of pyuria, bronchitis, and mastitis; 7 of these patients had well-marked fever for at least one day, and 8 of them became septic. The calculated morbidity of the mothers was 20 per cent. Of the children, 18.75 per cent. were stillborn; the fœtal morbidity is not stated. Induced labor was thought indicated when the true conjugate was 7 cm. or thereabouts. The most favorable period on the average, for induction of labor, was thirty-five weeks of gestation. Vertex presentations were most favorable, and of these 90.7 per cent. were born living. When the breech presented 61.5 per cent. were born living. The principal dangers of induced labor arose from injuries to the birth canal, and also to the child during operative delivery which terminated in induced labor. A further difficulty lies in the uncertainty of the actual period of gestation, and hence the difficulty in choosing accurately the most favorable time for the induction of labor. When the results obtained by induced labor are compared with the results of allowing the patient to go on to spontaneous labor, it is found that between two or three times as many living children are obtained after induced labor as after a spontaneous parturition. The children of induced labor, however, seem to possess but half the average strength of those spontaneously born. If children born by induced labor are followed for one year after birth it is observed that 20 per cent. less are living at the end of one year than of children born by other methods. The child's chance for life is diminished in accordance with the early period chosen for the interruption of pregnancy. Thus, before the thirty-fifth week the fœtal mortality is 10 per cent. greater than among children born by other methods.

Accidental Hemorrhage Complicating Pregnancy and Labor.—Wright (*Amer. Jour. Obst.*, November, 1906) reports the case of a multipara, seven months advanced, who while driving was taken with severe abdominal pains. Four days afterward she expelled spontaneously a dead child, with large clots. The placenta had been half discharged. When the uterus is exceedingly thin, and accidental hemorrhage occurs, it enlarges by the gradual formation of retained blood clot. In such a case this gradual enlargement of the uterus and its softness would furnish a valuable diagnostic sign. Wright considers pain and shock as especially valuable symptoms in this complication. He cites an obscure case of a multipara at eight months, in whom pain and shock developed. There was considerable abdominal tenderness, which disappeared after the bowels had moved freely, the patient ultimately making a good recovery. The treatment which he has found most successful consists in an enema of salt solution, sufficient morphine hypodermically to keep the patient perfectly quiet, strychnine $\frac{1}{30}$ grain, given not more than twice, keeping the patient's head as low as possible, and raising the foot of the bed. Especial attention should also be given to maintaining the heat of the body by external application.

The diagnosis of this condition is frequently especially difficult. It may be impossible before delivery to recognize clearly the condition. These patients rapidly become profoundly anemic; the uterus is distended in some to such an extent that it cannot be recognized by abdominal palpation. A condition of shock so severe may be present as to suggest a dangerous traumatism. When there is external as well as internal hemorrhage the diagnosis is more readily made. Retaining the blood within the uterus often produces effects difficult to recognize clearly. During the early stages of accidental hemorrhage blood clot stimulates the uterine muscle to contract, producing a condition of uterine tetanus. While this is present it is often impossible to recognize fœtal heart sounds or to map out the fœtus by palpation. If accidental hemorrhage follows severe traumatism the patient's shock becomes extreme. When hemorrhage without traumatism is present, the symptoms of collapse and loss of blood predominate. In the presence of considerable shock it is safest not to subject the patient to operation.

Suprarenal Extract in the Treatment of Osteomalacia.—Bossi (*Ctralb. Gynäk.*, 1907, No. 6) reports a case of twin pregnancy complicated by osteomalacia in which the disease of the pelvis was successfully treated by the administration of suprarenal extract. He further reports the case of a patient six months pregnant to whom the remedy was given by hypodermic injection. A practical cure of the condition was secured in twelve days. To test as far as possible the relation between disease or absence of the suprarenal bodies and osteomalacia, Bossi removed the right suprarenal body from a pregnant sheep. Osteomalacia developed immediately afterward.

Pregnancy after Sterilization by Atmokausis.—MEYER (*Ctralb. f. Gynäk.*, 1907, No. 6) reports the case of a multipara who had been treated by atmokausis for persistent uterine hemorrhage. The anterior wall of the uterus was greatly thinned and stretched, while the posterior wall seemed to have been greatly thickened by the treatment employed. The patient was about five months pregnant when vigorous bleeding came on. This was controlled by packing with iodoform gauze, and followed by operation. The case is interesting as showing that pregnancy is possible in cases in which the uterine cavity was supposed to have been obliterated by steam. Evidently the steam had attacked the posterior wall of the uterus and not the anterior. When pregnancy occurred and the ovum developed, that portion of the uterus not converted into cicatricial tissue became excessively distended and was, in fact, in danger of rupture. After atmokausis, not all patients so treated have obliteration of the uterine cavity. Fuchs in 18 of these cases observed obliteration in 11.

Multiple Sclerosis and Pregnancy.—WAGNER (*Ctralb. f. Gynäk.*, 1907, No. 6) reports the case of a primipara, aged thirty years, who forty-eight hours before admission to the hospital was attacked by cramps, which gradually developed into eclampsia. Paroxysms began in the region supplied by the right facial nerve, thence extending into the left upper extremity. The uterus was emptied under ether, and treatment addressed to the eclamptic condition was carried out. It proved, however, unsuccessful, the patient's strength failing until death occurred. At autopsy, multiple sclerosis of the brain and cord was present. The interesting question arises as to whether the convulsions depended upon the process from the brain and cord, or whether they were of the usual eclamptic variety, the lesions of the nervous system having nothing to do with the convulsions.

Cesarean Section for an Unusual Indication.—WECHSBERG (*Ctralb. f. Gynäk.*, 1907, No. 6) reports the case of a patient who came to the hospital in a pregnant condition, having a vesicovaginal fistula, the result of a previous difficult forceps delivery. After this the child died half an hour following birth. Repeated attempts had been made by operation to close the fistula, and this operation had been successful. During the last of her treatment the patient was found to be pregnant. After thorough examination, it was decided that delivery through the vagina would undoubtedly injure the urethra, and probably result in the formation of a fistula larger than the original. To avoid this, the patient was successfully delivered by Cesarean section.

GYNECOLOGY.

UNDER THE CHARGE OF

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Spontaneous Resection of the Tubes.—GUICCIARDI (*Ginecologia; Zentralbl. f. Gyn.*, 1906, No. 47, p. 1319) reports 5 cases of torsion of the Fallopian tube found in 1041 laparotomies and 3 in which spontaneous amputation had resulted. In the most typical a nullipara, aged forty-nine years, had a right sactosalpinx confined to the distal end. At a point an inch from the uterine cornu the tube was entirely separated, the ends being an inch apart. Guiceiardi believes that the mechanical factors in producing this condition are gradual elongation, and torsion or constriction of the tube, especially if it be dilated at the distal end (ampullary sactosalpinx).

Movable Kidneys in Women.—HEIDENHAIN (*Therap. Monatshefte*, 1906, No. 2, p. 1320) adheres to the theory of Wolkow and Delitzin with regard to the presence of "paravertebral niches," the normal position of the kidneys being maintained only when these are of a certain shape. In cases of movable right kidney the niche is notably shallow. The maintenance of the intra-abdominal equilibrium is most necessary to maintain the organs in their normal position; and this is secured through the elasticity of the abdominal walls and viscera. In operating it is not enough to fix the kidney in its normal position. Heidenhain prefers Schede's method.

Operations for Diseased Adnexa.—In discussing this subject before the Dresden Gynecological Society (*Zentralbl. f. Gyn.*, 1906, No. 49, p. 1352, LEOPOLD referred to a previous communication by Osterloh in which he reported 111 operations, in 52 of which drainage was employed, 10 of the latter cases having subsequently intestinal fistulæ, 2 of which resulted fatally. Leopold himself lost 2 per cent. of his last 150 cases, and drained only 9, in 4 of which intestinal fistulæ developed, 2 healing spontaneously. One of the patients in whose case drainage was employed died. Leopold's inference was that it is better to dispense entirely with gauze drainage. His plan is after walling off the field of operation to aspirate thoroughly a pyosalpinx or ovarian abscess, to clamp the opening made by the needle, and then carefully to enucleate the sac, sponging away pus as it appears. Under these conditions drainage is superfluous even in the worst cases, unless it is impossible to check oozing, when gauze is used, not as a drain, but as a tampon. In the discussion which followed Weindler affirmed that drainage was an unfortunate necessity, indicated only when large dead spaces were left. Schobach believed in drainage per vaginam only in recent pus cases of a doubtful character or when large raw surfaces existed. He used xeroform gauze, the drain being removed in five days. He had lost 1 out of 42 patients thus treated.

Gonococcic Peritonitis.—Leopold (*Zentralbl. f. Gyn.*, 1906, No. 49, p. 1354) reported a successful case of abdominal section for puerperal peritonitis, being the first one in his clinic in which gonococci had been found in the peritoneal exudate. The operation was performed eighteen hours after the inception of fever, the cavity being flushed daily with normal saline solution through three tubes—one in either groin and the other inserted through Douglas' pouch. The first two were left *in situ* for three weeks and the other for two weeks longer. Extensive granulation of the wounds occurred, but these eventually healed after the use of the sharp spoon and the patient made a good recovery.

Uterine Fibroid Complicated with Cancer.—Bland Sutton (*Jour. of Obstet. and Gyn. of Brit. Empire*, 1906, No. 7), in a series of 500 cases, met with only 8 in which there was accompanying adenocarcinoma of the corpus uteri; hence his inference that the presence of the benign neoplasm does not predispose the patient to the development of malignant disease. In his experience this complication is most common in unmarried or sterile women between the ages of fifty and sixty years. In 2 of the 500 cases primary cancer of the Fallopian tube was found.

Piquard (*Ann. de gyn. et d'obstét.; Zentralbl. f. Gyn.*, 1906, No. 49, p. 1368) bases his deductions upon 1000 cases of uterine fibromyoma, in 15 of which cancer of the corpus uteri was found. He infers that the chronic metritis induced by the presence of a fibroid predisposes to the development of cancer. Piquard was able to collect 179 cases in all from the literature. In this connection he reports 45 cases in which epitheliomatous degeneration of a fibroid occurred, though the majority of these were secondary to malignant disease of the endometrium. Primary cancerous degeneration of a fibromyoma was noted in only 24 cases, which are explained on the theory of preëxisting epithelial cells, possibly of embryonal origin, derived from the Wolffian bodies or Müller's ducts. The explanation that inflammation of the endometrium covering the fibroid leads to proliferation and invasion of the neoplasms by glands that subsequently degenerate seems more plausible.

Secondary Ovarian Tumors.—Amann (*Münch. med. Woch.; Zentralbl. f. Gyn.*, 1906, No. 49, p. 1365), from a study of 18 cases, recognizes three varieties of secondary cancer of the ovary: (1) Œdematous fibroma, with epithelial foci; (2) adenocarcinoma with subsequent cystic degeneration; (3) ovarian cystoma with discrete scirrhous foci.

In the first class the cancerous cells are distributed in the form of columns, which penetrate the fibrous tissue and often undergo colloid degeneration. Histologically they frequently give the impression of being endotheliomas. The hyperplasia of connective tissue represents a reaction of the ovary against the invasion of the neoplastic cells. This condition was noted in cases of cancer of the stomach and other viscera in which the ovaries were macroscopically unchanged.

It is not always possible, according to Amann, to prove the secondary origin of the ovarian neoplasm, especially when a small cancerous nodule in the stomach is associated with a large malignant ovarian tumor, except for the well-known fact that metastases in the stomach do not occur in connection with cancer of the ovary.

As regards the second variety, he states that while primary adenocarcinoma may develop in the ovary, double colloid cancer of both ovaries is rare, though it is quite common as a metastasis of gastric or intestinal cancer. In the third class secondary infection is possible, but rapid growth of pre-existing ovarian neoplasms in connection with long-standing cancer of the stomach and the presence of areas of œdematous connective tissue furnish evidence of metastasis. Infection may occur through implantation of detached cancerous masses on the surface of the ovary (!) or, if the original neoplasm in the wall of the stomach or intestine has not perforated it by way of the lymphatics.

The practical deduction is that in all cases of probable malignant disease of the ovaries the stomach should be carefully examined. In the event of an explorative incision being made, the viscera should be thoroughly palpated, since the primary tumor may be so small that it may even escape attention at the autopsy.

Resection of the Urete .—Zweifel reported a case before the Leipzig Obstetrical Society (*Zentralbl. f. Gyn.*, 1906, No. 43) in which he resected 5 cm. of the left ureter in connection with radical abdominal hysterectomy for cancer of the uterus, the proximal end being successfully sutured into the bladder. Of four similar operations in his clinic three were successful. In the discussion which followed Franz reported 17 cases of ureter resection, all but one in connection with extirpation of the cancerous uterus. Five patients succumbed to the operation. Of the twelve others (in two both ureters were implanted in the bladder) only one failed. Freund believed that when it was necessary to make extensive resection of the ureter high up it was better to avoid implanting the proximal end in the rectum or abdominal wound, or extirpation of the kidney, by uniting the end of the ureter with that of the Fallopian tube (uretero-salpingo-cystotomy). He had practised this method in dogs with good results. It is necessary to make a uterovaginal fistula afterward.

Ovarian Cyst with Chorio-epithelioma Metastasis.—Schmaus (*Beit. z. Geb. u. Gyn.*, 1906, Band x, Heft 2) reports a case of ovariotomy for adenocarcinoma of the ovary, in which the patient developed, a year later, nodules in the peritoneum and omentum which presented the histological appearance of chorio-epithelioma. He regards these as metastases rather than primary neoplasms, because of the fact that the origina cyst contained cell-formations resembling the secondary growths.

OTOLOGY.

UNDER THE CHARGE OF

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The Results Obtained from the Radical Operation for Chronic Purulent Otitis Media.—Edward Bradford Dench (*Annals of Otology, Rhinology, and Laryngology*, September, 1906) is of the opinion that, in the matter of permanent relief of the otorrhœa, the results obtained by this operation are not as flattering as had been hoped when the procedure was first suggested. Out of 193 cases, there were 131 cures, 29 with slight discharge, 5 with profuse discharge, 2 were still under treatment, there were 6 fatal cases, and 20 cases in which the result was unknown. Of the 6 deaths, 2 patients died of pneumonia, 2 of meningitis, 1 of cerebral abscess, and 1 of cerebellar abscess complicated by sarcoma of the auditory nerve. In no instance could death be attributed either directly or indirectly to the operation. Including the cases operated upon several years ago, and followed carefully, the results have been most satisfactory. Even when a small amount of discharge has persisted for weeks or even months after the operation, this discharge has in almost every instance disappeared absolutely, without the necessity for secondary interference, under simple measures of cleanliness.

It is not uncommon to have the patients return, at intervals of from four months to one or two years, complaining of some discharge from the external auditory canal, the entire cavity being filled with a mass of desquamated epithelium, upon the removal of which a small amount of pus may be found. If this accumulation has remained in the canal for a considerable period of time, desquamation of the epithelial lining of the cavity may have taken place either completely or over certain areas. If the cavity is simply sterilized with an alcoholic solution of bichloride of mercury, and then dusted with some bland, non-irritating powder, such as boric acid, xeroform, zinc oxide, or, in fact, any sterile powder, the cavity becomes dry, the apparent relapse in many of the cases being due to the fact that the integument lining the middle-ear cavity and the mastoid cells, is improperly nourished, the integument forming the lining of a blind pouch being subjected to increased temperature, increased moisture, and deprived of light and of air, which are essential to the proper nutrition of the normal skin; this tegumentary lining, moreover, is applied closely to the bony walls of the cavity, with very little connective tissue substance intervening.

These attacks of desquamation occur less and less frequently, because the integument gradually adapts itself to its new habitat, and conforms itself to its anomalous position; they should not be looked upon as a recurrence of the otitis, since they are easily relieved by the removal of the walls of the cavity.

Out of 111 cases, in which the hearing records were kept, the hearing after the operation was good in 99, was fair in 9, and was bad in 3. Out of the last 95 cases operated on, facial paralysis occurred in 4 instances, and the function was ultimately restored in all.

As regards the technique of the operation, the author is in favor of the employment of large skin grafts applied, preferably, through the posterior opening, on account of the shortening of the after treatment.

Arteriosclerosis of the Labyrinth and Acoustic Centres.—E. Escat (*Annales des maladies de l'oreille et du larynx*, 1906, xxxii, 325) gives a most interesting paper that furnishes food for a great deal of thought on the part of otologists. He considers it astonishing that a study of the localization of arteriosclerosis in the ear has not sooner found a place in otological pathology. He chides the profession for not having better applied general medical knowledge to the problems of special organs, points out some of the difficulties in the way of studying the pathology of the internal ear, and sets forth the proposition that "in the same way that a cortical centre of the brain, a pyramid of the kidney, an hepatic or splenic lobule, or a retina may suffer from arterial spasm or the cutting off of its nourishment, so may the labyrinth. The internal auditory artery, as well as the iliac or coronary, may suffer from hypertension and endarteritis. The study of otitic arteriosclerosis is exactly homologous with that of the eye."

A schematic drawing is presented to show the distribution of nutritive arteries to the labyrinth and to explain how, by the anatomical disposition of the arteries, arteriosclerosis may be limited to a single department of the auditory apparatus or may involve it extensively. He discusses the pathology of arteriosclerosis and points out that "the process would act the same on the labyrinth as on the acoustic centres, because, like the brain, it is but a peripheral expansion of the brain. The paroxysmal form of the syndrome of Ménière, with complete remissions, shows a crippling of the labyrinth by spasm of the internal auditory. The apoplectic form of the same syndrome with intralabyrinthine hemorrhage, is similar to rupture of an artery in cerebral hemorrhage or in epistaxis. Now, if degeneration of the coats of the terminals of the internal auditory causes hemorrhage it is also certain that endarteritis of the same vessels will lead to slow atrophy of the organ. The lesion may occur in the vestibule, semicircular canals, termination of the vestibular nerve, or the ganglion of Scarpa."

Clinically he divides the labyrinthine troubles caused by arteriosclerosis into three types: (1) Deafness without vertigo; (2) vertigo without deafness, and (3) deafness and vertigo associated.

"Arteriosclerotic deafness comes on slowly, insidiously, and is progressive; general arteriosclerosis may or may not have become evident; it is generally worse on one side than on the other; there is less impairment of perception of the low than of the high tones; bone conduction is shortened; the patient hears better in silence than in a noise. Objective examination may show but little evidence of change in the membrana tympani; occasionally there is a sclerotic white ring about the periphery, analogous to the arcus senilis of the cornea, but little importance can be attached to this. There is usually no evidence of ankylosis of the ossicles and nasopharyngeal examinations fail to disclose any sufficient abnormality to explain the impaired hearing. The diagnosis then rests upon the existence of a gradually progressive deafness, presenting the characteristics of a lesion of the perceptive apparatus, and, general signs of arteriosclerosis, such as hypertension and induration of the peripheral arteries. When the last-named elements are lacking the diagnosis is reduced to a presumption and must await confirmation by later developments.

"Deafness without vertigo will be produced by arteriosclerosis when the lesion involves the vessels or centres supplying the cochlea only, and leaves those of the vestibule unimpaired; the four principal foci, where this may occur, are the cochlear artery proper, the cochlear nuclei in the pons, in the posterior arm of the capsule, or the temporal branch of the sylvian artery. The slowly progressing form is due to obliterative endarteritis, the rapid form to emboli.

"Vertigo without deafness is capable of being produced by an arteriosclerosis in a region which affects the vestibular vessels or centres which control equilibrium, without coincident involvement of the cochlear circulation.

"Deafness and vertigo associated is much more commonly recognized, probably because it usually presents the syndrome of Ménière so clearly; that is, tinnitus and nausea are added to the above symptoms. Here the lesion is apt to be an arteriosclerosis of the internal artery itself and a consequent interference with both the nuclear and vestibular vessels. Labyrinthine hemorrhage or embolism will produce the symptoms rapidly, while a progressive sclerosis of the internal auditory artery causes a gradual development of the syndrome. Likewise, lesions in the cortex or pons that involve centres controlling the equilibrium will produce the complex type of the disease.

"The two conditions especially to be borne in mind when making a differential diagnosis are hereditary or secondary syphilitic labyrinthitis and otosclerosis. The hemorrhagic labyrinthitis occasionally occurring in chronic kidney disease may at times be difficult to differentiate."

Escat gives some plates to illustrate the pathological changes exhibited in an autopsy and urges the importance of everyone having the opportunity of making careful postmortem examinations in suspected cases. If Escat's reasoning be correct, we have a satisfactory explanation for many cases of deafness that have heretofore seemed very obscure. The prognosis and treatment is naturally that of general arteriosclerosis. Considering the symptoms separately, it may be said that deafness is the least curable, the tinnitus may become permanent, and the vertigo generally disappears. The best form of treatment Escat has found consists in the administration of hydrobromic acid in increasing doses while following the dietary of arteriosclerosis.

PATHOLOGY AND BACTERIOLOGY.

UNDER THE CHARGE OF

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Experimental Anemias in the Rabbit.—C. H. Bunting (*Jour. Exp. Med.*, 1906, viii, 625) has made an exceedingly careful study of the changes in the blood and bone-marrow of rabbits which have been subjected to injections of the hemolytic substances, ricin and saponin. The work was controlled by a study of the anemias produced by bleeding and by injections of substances acting differently from ricin and saponin. The results of these experiments are highly suggestive and aid to a clearer understanding of the difference between a secondary and a primary anemia. Ricin has a marked toxic action on the blood elements, and when injected intravenously even in small doses in rabbits is quickly fatal. The destructive action on the leukocytes is evidenced by a rapid leukopenia, followed by a sharp reaction which reaches a considerable height at the end of twenty-four hours. During the stage of leukocytosis the white cells show signs of injury. With acute ricin poisoning the red cells are not greatly reduced in numbers, but in the circulation there appear enormous numbers of nucleated red blood corpuscles. The condition found in the bone-marrow serves to explain these changes. In the normal rabbit the cells of the bone-marrow are arranged in more or less definite groups which Bunting terms the erythrogenic and leukoblastic centres. The centres of the erythrogenic patches are composed of indifferent myeloblasts, while in the surrounding layers are megaloblasts, intermediate red cells, and normoblasts, and about the extreme periphery mature red blood cells. The white-cell centres are formed by layers of indifferent myeloblasts, myelocytes, and adult polymorphonuclear leukocytes. The marrows of the rabbits dying from ricin poisoning show marked depletion of many cells, and pycnosis and fragmentation of the remaining cells which go to form the proliferating centres for the red and white cells. This goes to show that ricin has a profound toxic action not only upon the cells of the circulation, but upon the cells of the bone-marrow. Here lies the essential difference between the secondary and primary anemias.

Rabbits bled on successive days developed a secondary anemia of moderate grade, with occasional nucleated red blood corpuscles in the circulation, but the marrow cells showed no injury. There was a proliferation of the erythrogenic centres in the normal manner.

Further experiments showed that by repeated injections of ricin a moderate grade of chronic anemia could be brought about in which the blood picture, except for a leukocytosis, resembled very closely that of pernicious anemia in man. Nucleated red blood corpuscles, usually normoblasts but often megaloblasts, were constantly present; macrocytes and microcytes formed a considerable percentage of the cells; and as the anemia progressed there appeared polychromatophilia and at times basophilia. By using saponin a chronic anemia could be produced which was even more strikingly like pernicious anemia, since there was a leukopenia with relative increase of the lymphocytes. In these cases the circulating blood often showed as many as 5000 to 10,000 nucleated red blood cells per cubic millimeter, 100 to 300 of which were megaloblasts. The lesions, confined almost exclusively to the bone-marrow in these cases of saponin poisoning, were of particular interest. Besides the injury to the cells there were large hemorrhages. Later, a great increase in connective tissue was found, which diminished greatly the space available for the hemopoietic tissue. So marked was this sclerosis that it was difficult to explain how the red count of 2,000,000 to 3,000,000 could be maintained. The explanation was found, however, in the fact that in the peripheral sinuses of the spleen there appeared to be a vicarious formation of red blood cells. The peripheral sinuses of the spleen were distended and crowded with cells, chiefly of the erythrogenetic series, though there were also megalokaryocytes and leukocytes. The nucleated red blood cells were grouped much as they are in the marrow and showed numerous mitotic figures.

Bunting concludes that red-cell crises in the circulating blood are an expression of injury to the bone-marrow. When the mature erythrocytes about the periphery of the erythrogenic centres are depleted by hemorrhage, or the action of a circulating toxin, the bone-marrow reacts with nucleated cells. In the first case the reaction is much less marked than in the second. Following toxic injury to the marrow with destruction of the peripheral cells of the erythrogenic groups, there is established an atypical formation of erythrocytes resulting in pathological forms. When the marrow is extensively injured the spleen may take on the hemopoietic function. With subcutaneous injections of the hemolytic toxin the absorption is so slow that the toxin is apparently saturated by cells in the circulation and does not reach the marrow in sufficient quantity to injure it. Under those conditions there is blood destruction, but no nucleated red-cell crises in the circulation, and the marrow picture is one of hyperplasia, such as is seen after hemorrhage, that is, the marrow of a secondary anemia.

Studies upon Acromegaly, with Special Reference to the Connection between Acromegaly and Tumors of the Hypophysis.—Three hypotheses have been advanced to explain the connection between acromegaly and tumors of the hypophysis: (1) That the function of the hypophysis is partially or entirely destroyed; (2) that the function of the hypophysis is increased, and (3) that the acromegaly is primarily due to a disturbance of metabolism and that the hypophysis tumor is a symptom of the disease. CAGNETTO (*Virch. Arch.*, 1907, clxxxvii, 197) has already studied 2 cases of hypophysis tumor, in 1 of which, without acromegaly, the hypophysis was entirely destroyed by a sarcoma arising from another tissue; he now reports 3 more cases, describes enlarged hypophyses in 2 pregnant women. Acromegaly was present in the first 3 cases. A study of these cases and a review of the literature seem to throw much doubt upon the hypophysis theory of acromegaly, and especially upon the theory of "hypepitiutarismus" (hypersecretion of the hypophysis). Acromegaly occurs without hyperplasia of the glandular lobes of the organ; acromegaly may occur in association with a tumor free from functionating elements (chromophile cells); tumors of the hypophysis occur with numerous functionating cells in which there is no associated acromegaly.

Changes in the Bloodvessels and Organs brought about by Injections of Adrenalin Preparations.—The alterations in the walls of the arteries of rabbits, described first by Josué, in 1903, following injections of adrenalin, have now been produced by various observers and described in detail. They consist briefly of a necrosis in the middle coat followed by matting together of the elastic fibers that later on break up in fragments. Inflammatory reaction follows with infiltration by lymphocytes and the formation of giant cells. Subsequently there is calcification with thickening of the intima or aneurysmal bulgings. Recently v. KOVANJE (*Deut. med. Woch.*, 1906, No. 17) and BOVERI (*Deut. med. Woch.*, 1906, No. 22) have stated that the effects of the adrenalin injections may be greatly modified or partially neutralized by simultaneous injections of iodine preparations. Biland (*Deut. Arch. f. klin. Med.*, 1906, lxxxvii, 413) has repeated these experiments and comes to a different conclusion. Eighteen rabbits were injected subcutaneously with adrenalin hydrochloride. Of these, eight were also given doses of potassium iodide. One rabbit received thirteen injections of the iodide. The injections of the iodide did not seem to neutralize in the least the action of the adrenalin. Indeed, in the rabbits which received both potassium iodide and adrenalin it was found that the lesions of the bloodvessels were more marked than in the animals which received the adrenalin alone. Further studies were made to determine the effect which the adrenalin might have upon the blood pressure. The blood pressure of nine injected animals varied between 90 and 130 mm. Hg.; in the control animals the pressure varied between 85 and 110 mm. Hg. There was no parallelism between the height of the blood pressure and the extent of the vascular lesions. Immediately after the first injection of adrenalin the blood pressure rose to from 180 to 222 mm. Hg. Following this there was a rapid fall within five or seven minutes to normal. The effect of subsequent injections, even in very large doses, was much less marked, and a second injection, given before the blood pressure curve following the first had reached normal, produced no effect upon the regular descent. Though Biland could not determine that an increase of blood pressure played any part in the production of the vascular lesions, he could find evidence to show that the action of the adrenalin was dependent upon a toxic necrosis of the coats of the bloodvessels. In the kidneys more or less diffuse or localized areas of necrosis were encountered. They involved principally the epithelium of the tubuli contorti, and more rarely the loops of Henle. Occasionally there were calcium deposits, oftenest in the lumen of the tubuli contorti and tubuli recti. Not only are there evidences of an intoxication of the cells of the kidney, but toxins are eliminated in the urine. The urine from rabbits treated with adrenalin was highly toxic, and in doses of 0.5 to 1 cm. it was fatal to healthy animals, which died in tetanic convulsions. The injections of toxic urine caused a marked decrease in blood pressure, and in other ways acted quite differently from adrenalin, so that Biland concludes that its injurious effect is not due to the elimination of adrenalin by the kidneys.

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